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THE BRITISH JOURNAL OF CHILDREN'S DISEASES.

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THE

BRITISH JOURNAL

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CHILDREN'S DISEASES.

VOL. XX. APRIL—JUNE, 1923. NOS. 232-234.

Original Articles.

THE TREATMENT OF ACUTE OTITIS MEDIA IN CHILDREN.

By T. B. LAYTON, D.S.O., M.S.Lond., F.R.C.S.,

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IF the story of present-day medicine ever be faithfully written, one of the saddest chapters in it will be that which deals with chronic discharge from the ears in children. Sometimes this starts as a slight complaint which mothers and school matrons regard as trivial. Sometimes it arises as a minor complication of a specific fever or other serious illness. Instead of getting well, it becomes firmly established within the ear. The child begins to go deaf and ceases to hear clearly all that is said to him, and at an important stage of his educational career one of the portals to the developing mind is partially cut off. Then follows a loss of attention, or a failure to develop that voluntary attention which is an essential to all good work. Or the youth becomes introspective, keeping himself to himself, suspicious of all that surround him, shunning them and not mixing with the life of the day, so that his whole character develops along some line unsuited to the part he has to play. All this is only one side of the picture. So long as any active disease remains the person is as one who lives over a bomb, and no one can tell whether this is a live one or not. At any hour of any day the explosion may cause death in a few hours from meningitis or from cerebral abscess. Or it may be followed by an illness, prolonged and serious, from which the patient only

recovers after the pain of a major operation and of a lengthy convalescence.

And yet when the drumheads of adults are examined it is surprising how often signs of disease are seen. Otitis media must have occurred in many persons who have no knowledge that they ever suffered from it. This can only mean that at some stage the disease has a tendency to cure itself. From it we may infer that at this stage it should be possible to assist Nature to cure the vast majority of cases, so that the terrible results of running ears outlined above would not happen. Such a stage is probably present in the first few days after the onset of the disease. Difficult as is the treatment of chronic otitis media, and discouraging as it is to all who are engaged in it, the treatment of acute otitis media is relatively simple, and, given fair opportunities to apply it, is stimulating in its success.

It is the purpose of this paper to detail the symptoms of acute otitis media, to describe the treatment which the writer has found useful, and to indicate the ways in which this may be carried out.

The objective symptoms of acute otitis media are found in the drumhead, and in the drumhead only; an examination of this is therefore essential in every case.

The earliest evidence of inflammation in the middle ear is a flash of dilated blood-vessels running upwards and backwards from the neck of the malleus. Like a bundle of ribbons tied at one end they stream across the flaccid part of the membrane to lose themselves in the contiguous skin of the meatus, while one that runs down the handle of the malleus looks like a limply hanging solitary streamer that has not been caught by the wind.

In the next stage there is a diffuse pinkness. In this, as in subsequent stages, the disease may be local or general, but the former is more common. The signs are usually confined to the upper and back part of the drumhead above its posterior fold. When a diffuse pinkness extends over the whole drumhead there is no difficulty in recognising it, but when the postero-superior quadrant alone is involved, there is often considerable doubt as to whether it is the drumhead or the meatal wall that is reddened. The difficulty is due to three things: First, the anatomical one that in this quadrant, even in the normal ear, it is hard to say where the meatus ends and drumhead starts. If one looks at the anterior and the inferior parts, one can see a definite white line at the junction of meatal skin and drumhead, but in this postero-superior quadrant the pink of the one fades through a white opacity into the greyish translucency of the other. The next cause of difficulty is due to the ease with which the skin in this area becomes injected from any slight irritation, the pressure of a small

piece of wax, or the mechanical irritation of the water or the instrument used to remove this, and with this injection may even be associated a dilatation of the blood-vessel running down the handle of the malleus. The third reason is that the redness of early inflammation of the drumhead soon spreads to the neighbouring skin of the meatus. Hence, with the indefinite anatomical boundary it is hard to say whether the redness is limited to the skin or is a redness of the drumhead spreading on to this. The diagnosis depends chiefly upon the examination of the ear by noting carefully all the anatomical details of the membrane in their relation to the redness, and above all by a comparison with the other ear in a case of unilateral disease. In a doubtful case other small points may just turn the balance in favour of one lesion or the other. Thus, if the examination is made owing to the child's complaint of pain, the absence of any other cause of ear-ache and the fact that ear disease is the commonest cause of this will decide in favour of the middle as against the outer ear. Again, an examination of the throat may help, for with no sign of inflammation here it is easier to exclude the middle ear, while a careful look at the lymphoid tissue which runs down behind the posterior pillar of the fauces may show this to be swollen and red on the side where are the ear signs, when compared with the same structure on the other side of the pharynx.

Fortunately at this stage it is not of vast importance to decide at once. The simple treatment to be detailed later is suitable for either condition. The importance of the stage is that it affords a warning that this ear should be watched from day to day to see whether the condition is going to pass over into the stages now to be described.

The third may be called the stage of the "full" drumhead. At this stage an infiltration into the tissues has occurred, filling up the hollow spaces, but not yet causing convexity outwards. It is recognised by noting that the anatomical landmarks of the postero-superior quadrant are difficult to observe. The diffuse pinkness of the last stage is present or more marked so that it has become an intense redness. The determining line between drumhead and meatus is more difficult to define than ever—in fact it is seldom necessary to try, for nearly always the contiguous wall of the meatus is involved in the inflammation,—but in addition the posterior fold can barely be made out; the concavity between its lower margin and the posterior margin of the handle of the malleus has gone, as has also that above the posterior fold and the upper border of the tympanic margin. Into this area the short process of the malleus no longer juts, but may be seen merely as a white dot in a sea of red. This stage is important as a warning, not that the ear "should be watched from day to day," but that it must be watched from morning to night,

and then from night to morning again, in order that the information may at once be got if the condition has passed on from this to the next stage, at which prompt action is needed.

In this stage the drumhead is " bulged," the whole or part being convex towards the outer meatus of the ear. The convexity of the whole is more easy to observe, but is rarer, and in most cases the postero-superior quadrant alone is involved. There are no special physical signs whereby this condition can be recognised; it depends upon observing that the surface is convex towards the exterior. It is sometimes difficult to see this by monocular vision through an otoscope. When the condition is localised to the postero-superior quadrant there is the contrast between this and the rest of the drumhead, and a light reflex is present at the area of greatest convexity; when the whole drumhead is bulged the curving of dilated blood-vessels which radiate inwards from the periphery affords the best means of estimating the convexity.

Acute otitis media is not a disease by itself; it is a part of an infection of the upper respiratory tract, and general treatment is as important as local treatment. The child must be kept quiet, confined to the house, preferably in bed. The room must be warm but ventilated, the latter being more important than the former. A through ventilation must be maintained even though the mother fears she cannot keep the room warm with the window open. A cold current of air impinging on the ear or side of the head will increase pain and do harm; but the ear must be protected by the local means soon to be described, and not at the expense of fresh air in the room. Efficient ventilation is essential to good treatment in this as in all other diseases of the upper respiratory tract; and the want of it is, in the writer's opinion, an important factor in determining whether the other ear becomes involved or not. A brisk purge should always be given thoroughly to empty the gut, and for this castor oil is unrivalled. Let not the mother put one off by saying she gave the child a dose of liquorice two nights ago; if there is any fur on the tongue or any tendency to dryness of it the oil should be given just the same. Once the gut has been emptied, the bowels may be left to themselves for several days. It matters not what the child has to eat so long as he takes plenty to drink and takes it hot. I usually advise a treacle posset hot, of equal parts of milk and water, into which a dessert-spoonful of golden syrup has been stirred. This is easy for the mother to make, the child likes it, and if it is taken several times a day he is getting sufficient kilo-calories, even if he refuse all other food. This may be supplemented by hot sweet lemon drinks as often as the child likes. The sugar in each provides a foodstuff readily digested and absorbed from a disordered stomach. It also helps to stimulate secretion of

mucus in the upper respiratory tract, and it is this that is needed to combat the infection. To stimulate this secretion by general means is of more value than to apply locally any antiseptic, which can only act on a very small part of the mucous membrane. For this reason I urge the use of fluids, preferably hot, and for this alone I would give drugs. Most children will like to suck a tablet of chlorate of potash, and it is in this way, I believe, that an alkaline mixture acts. I am opposed to the use of aspirin or the salicylates ; they have no special action except to lower the temperature, and to leave the child weak, depressed and prostrate, with a lowered resistance during a period when active organisms are still present in the throat liable to cause a recurrence of the attack. If any drug be needed the good results can all be got by a few doses of liquor ammoniæ acetatis, without the bad after-effects of the aspirin. Lastly, an inhalation of Friar's balsam will be of value in a child old enough to use it ; or a steam kettle in a younger one if it be thought advisable. When this stimulation of mucus has been effected it is necessary to encourage the child to get rid of it, to provide him with a spittoon and to tell him to spit into this all that comes into the mouth, to teach him to blow the nose correctly. Let him obstruct one nostril with the finger while he leaves the other patent through which to blow the muco-pus out of the nasal organ, so that it does not go just down into the vestibule, from which it is sniffed up again into the fossa as soon as the handkerchief is removed. To teach this the mother should hold the handkerchief some inch or so away from the nose as a receptacle into which the secretion may be caught while she blocks the other nostril with the finger, and as she does this she can often aid by catching hold of long strings of muco-pus that come half out of the nose and would be inspired again were they not thus digitally removed. It is in this connection with the removal of mucus that a mouthwash is of value. As it clears the mucus away from around the teeth and gums and from the folds of the cheek, the glands again secrete and other mucus takes its place. The wash must therefore be pleasant and alkaline. Its antiseptic value is of secondary importance. Never must it be so strong that it hurts the mouth, or it may do more harm to the mucous membrane than to the microbes. For these reasons I use the following :

Lysol	1 part.
Glycerine . . .	50 parts.
Tincture of lavender .	*q.s.*
Water to the 100 parts.	

This is to be used with two or more parts of hot water frequently.

And for these reasons I do not order a gargle ; it is doubtful whether

there be any direct action of the antiseptic on the organisms living on the mucous membrane of the tonsils or the pharyngeal wall, and if there be, it is only in a small number of persons that the fluid with which they gargle touches these parts. On the other hand, every attempt to use a gargle throws into violent action all the muscles of the pharyngeal wall which move and hit the overlying inflamed mucosa. Thereby is violated the principle of the therapeutic value of rest. This is as important in the treatment of an inflammation of the mucous membrane of the throat as it is in that of the synovial membrane of the knee-joint.

This rest must be general as well as local, and all this treatment we have described should not be used if it disturbs the patient. The child, being a young animal, curls up and sleeps off an illness if relieved of pain; and if he will do this he should not be disturbed except to take his food or for some other essential reason.

The local treatment of acute otitis media may be summed up by saying that it consists in performing the operation of incision of the drumhead at the right moment. It should be the aim of the practitioner never to allow a drumhead to rupture spontaneously; to look upon an inflamed ear which, under his observation, has gone on untouched to the stage of discharge as something of a disgrace to himself, just as he would consider to be so a case of acute appendicitis which in these circumstances had gone on to general peritonitis. In many a case in which the drumhead bursts it heals again and the ear gets well, but there are many in which it does not do so. It is idle to discuss the proportion of the one to the other; conditions of practice give different men different impressions: one sees more of the tragedies, another of the recoveries. The point on which it is important to concentrate is that if this ideal were realised cases of acute mastoiditis would be few, the complications of meningitis and sinus thrombosis pathological curiosities, and chronic running ears of rare occurrence, instead of being the despair alike of the ear surgeon, the social worker, and of the school administrator.

Let it not be thought there is no other local treatment than this, that we have but to wait for the appropriate moment before doing anything to the ear at all. In the early stages we can relieve pain, we can render the external auditory meatus less highly infective in case a hole in the drumhead occur later, whether spontaneous or made by the surgeon, and we can apply to the outer surface of the drumhead some drops, whereby we hope to modify in part the changes that are occurring on its other side. We hope thereby and by the general treatment already mentioned to cause the attack to abate so that it never reaches the stage where an incision is needed. But nothing that we do must interfere with our power of examining the drumhead; once a clear view of this

has been obtained nothing must be done to obstruct it; to watch it day by day and almost hour by hour is the only way to know whether an operation will be needed, and the only way to get a good result.

It is for this reason that hot fomentations are so bad and should never be used. True, they relieve pain; in many a home they may be the easiest and most efficient way of doing this; but their disadvantages quite outweigh their usefulness, for it is almost impossible ever to see the drumhead once they have been used. If a practitioner watches the drumheads in his own cases he will soon learn not to use them. If he call in an otologist he has no right to ruin the chances of making an efficient examination and thereby of giving a good opinion. For the moisture of the foment gets into the meatus and causes the superficial epithelium to swell up, and entangles in this any little bits of wax that lie about, and this soft mass occludes entirely any view of the drumhead. To remove this while the meatus is tender with the child ill and in pain is impossible without an anæsthetic. The application therefore of foments reduces the treatment of otitis media to a tragic farce, in which the practitioner is joyfully going on in a neglectful mood of *laissez aller*, or is driven to give an anæsthetic that should have been unnecessary, and may have to be repeated in a few hours' time if the best is to be done for the patient.

The way to relieve pain is to apply heat to the ear and to the side of the head; but, as we have seen already, this must be dry heat. In hospitals and places where they keep sufficient apparatus this may be done with an electric coil, but in the home a rubber hot-water bottle half filled and covered with flannel is the most efficient. In his sleep, however, the patient may roll off the bottle and expose the ear to a cold current of air, and so a cap should be put on the head to cover up the ear. This may be one specially made of flannel with a tape to tie beneath the chin, or it may be improvised from some old shawl or soft flannel garment, or some woollen form of head-gear may be brought into use for the purpose. When no hot-water bottle is available a pad of wool may be tucked beneath the cap to supply the warmth, and this may be rendered more efficient by using thermogene wool or some such preparation. In addition drops should be placed within the ear. These should be antiseptic to sterilise the meatus, and that they may exert some influence upon the disease they should be warm and hygroscopic; for this last reason and that they may not swell up the epithelial *débris* and so obscure the view they should contain no water. Some use the glycerinum acidi carbolici B.P., but this has a strength of 20 per cent. of carbolic acid, and though harmless in many ears occasionally it may cause irritation and raise a suspicion of mastoid disease, or make the diagnosis of this much harder when the suspicion is aroused from some other reason.

These, then, need dilution with plain glycerine, but as glycerine is somewhat viscid and hard to pour into the ear some spirit (rectified or methylated) may also be added. We have as stock drops, then, the following prescription :

Glycerine of carbolic acid . . .	1 part.
Methylated (or rectified) spirit . .	1 part.
Glycerine	2 parts.

Care must be taken that these be not put in the ear too hot or the skin of the meatus will be burnt, not only causing a painful lesion, but preventing absolutely any accurate appreciation of the cause of the otitis media, and possibly making a mastoiditis impossible to recognise.

Short of burning, drops which are too hot will cause a giddiness so intense that the patient cannot stand them again. It is well then to warn the parent to warm the drops in a teaspoon, and to test this spoon upon the tender skin of the front of the forearm or over the back of the proximal phalanx of the little finger. If it be too hot for this the drops are too hot for the ear, but if it gives a pleasing warmth to the skin they will be all right. It is easy to scoff at ear drops as some do, and to say they can have no effect upon the course of a disease in a structure from which they are separated by the fibrous layer of the drumhead. It is, of course, a matter impossible of proof, but who can say they will not turn the scale from time to time in favour of resolution ? And even if they only serve to relieve the pain and to apply an antiseptic to the meatus they have served a purpose.

When, then, is all this treatment insufficient, and when should we incise the drumhead ? In every case in which this has been observed to be bulged. The cases clinically fall into two classes. First, those where the convexity is seen at the first time the ear is observed ; in every case this operation should be done at once unless the severity of the attack is already passed, the pain has gone and the temperature has fallen. Then for a few hours only, but not overnight, it is permissible to wait, and to see if the swelling is subsiding. In a few—a very few—this may occur. In the vast majority if left the drumhead will burst. Second, those which have been watched from day to day and the inflammation has gone through all its stages. In these cases there is no need to wait ; the operation should be done at once. In some of this class it is not necessary to wait for the drumhead to bulge, but it should be incised merely for fullness. If the child has been for more than a day in pain, if it has spent one night, and perhaps two, restless and crying, awake and keeping the mother awake, so that she is tired out and unable to give her full attention to the child, if the temperature remains up, if the tongue still is furred, and the drumhead has progressed from redness to a fullness, then it is

unnecessary to await the completion of the bulge ; the incision should be done at once with a view to preventing mastoiditis and to relieving pain, thus giving the child a sleep from which he may arise refreshed and convalescent.

Even in adults I have found no local anæsthetic thoroughly reliable, so in children I will not discuss it. The operation can be done without an anæsthetic, but I do not advise it. One quick stab and one only is possible. If it miss, nothing further can be done ; and, even if successful, it is more likely to be a puncture than incision. The operation should be done deliberately and with precision after cleaning up the meatus and viewing again the condition of the drumhead to confirm the former observation. For this reason the shorter anæsthetics of ethyl chloride or gas should not be used ; the latter has the further disadvantage of causing blueness, which changes all the picture of the drumhead and makes it necessary to pick out the landmarks afresh. Ether or pure chloroform given by the most open method are the best ; the patient need only be deeply enough under not to move. The writer always uses reflected light caught from a lamp or from the window, but many prefer an electric otoscope. The incision should be made where the drumhead is bulged along the full length of its greatest convexity. There is no special site where it should be made, for the convexity varies with every case, but it is best made from below upwards. Where a swelling also exists on the posterior wall of the meatus this also should be opened by a cut going forward to meet the last at its upper end. Through the wound pus is seldom seen ; sometimes muco-pus exudes, but usually blood only flows, to be followed in forty-eight hours by a purulent discharge. During this time nothing need be done except to tie a pad of wool over the ear, and to continue with the other treatment.

So far we have only considered the objective physical signs upon which the diagnosis is made. This has been done because they can only be seen by using the aural speculum, because without its use efficient treatment cannot be carried out, and because the employment of this instrument is not as common as it should be. There must be, however, certain symptoms which will draw attention to the ear and warrant such an examination. These are the subjective and general symptoms of the disease, which are pain and a raised temperature. Pain is the most important of all, and in every child who complains of pain the ear should be examined. We want to train the medical profession to look upon the instillation of drops without examination into the ear of a child who complains of pain as being as great a crime as we now consider the administration without examining the abdomen of a dose of castor oil to a child who complains of pain in the belly. Acute otitis media through its

complications may be as serious as acute appendicitis. Years after the onset of otorrhœa the adult dies in a few hours from a meningitis that might have been saved had a proper treatment been instituted on the day the child complained of pain. Neglect is not brought home as it is to the doctor who on the fourth day learns that the child he is treating for a "flatulent colic" has general peritonitis, but the responsibility for that person's death is just the same.

Not everyone who has an inflamed ear complains of pain. In the specific fevers otitis media often passes on to rupture without anything to draw attention to the ears. In neither measles nor scarlet fever is earache common; in the latter a slight pyrexia may occur in the apyrexial stage; in the former the otitis more often occurs before the temperature has subsided. In these diseases there are a number of cases in which the disease is not recognised till the drumhead is already burst. The same may be true of other acute diseases in childhood. This does not mean we must give up trying to see the ears before they burst; rather it means we must the more be on the alert to examine them. They should therefore always be examined in any case of sore throat; and especially after the operation for tonsils and adenoids. It is well also to do so when a child has a pyrexia of otherwise unexplained origin. Lastly the other ear should always be examined in any case where one ear is inflamed.

Let it not be thought that it is always easy to see a child's ear. Even when there is no pain in it the examination is often difficult; when the child is crying with pain it is more so. The chief difficulty is in the child himself, or rather in the parents, for almost without exception the child who fights and struggles against examination is accompanied by a mother, who weakly gives way to all the child's whims, and then suddenly threatens him with severe penalties if he is not, as she puts it, "good" during the examination. Mothers, nurses, matrons of schools or ladies of care committees should also be vigorously suppressed when saying "Come along, dear! the doctor is not going to hurt you," thus suggesting to the child that he is about to do so, and perhaps lying to him. The examination may be a little painful, and to lie to a child is the worst way possible to get his confidence. It is in getting this confidence that half the art of seeing the child's ear lies. For this reason I have given up sitting the child in a chair; the very paraphernalia of the rite of examination frighten him; the speculum, the darkened room, the bright electric light together give some suggestion of an awful ceremony which sends a terror into the heart of the child. If he is not made to sit down the ceremony is less formal; to stand the bigger child between the knees or to sit the smaller on them gives a confidence while you talk and examine the sound ear. For the same reason it is often well to dispense with the

more efficient electric light in a darkened room, and at first to use some less strong ray of light than comes in from the window. The frontal mirror is seldom feared; it is rather a source of wonder, of interest, or even of amusement. When you have the child's confidence so that you can see into the ear you have still to see the drumhead, and here are two difficulties—wax and the narrowness of the meatus. To remove wax from a child's ear when the drumhead beneath is inflamed is the hardest operation in all otology. The syringe is valueless, the child will never stand it, and if he does it will only be at the expense of such pain and tears that a subsequent look at the drumhead is impossible. Very slowly, very carefully, and very gently with a fine wax curette it may be removed in many cases; but there remain some in which we must confess to failure and admit that it is impossible to examine the ear. By trying his best it should be the aim of every practitioner to make this number minimal. The same thing results from the extreme narrowness of the infant's meatus: the books on anatomy annoyingly state that the drumhead of the newborn babe is practically as large as it is in the adult; they omit to add, or the student fails to notice, that this does not apply to the lumen of the external auditory meatus. I am not sure that I have ever given a good opinion on the drumhead of a child under two without the aid of a general anæsthetic. The solution to all these cases in which the drumhead cannot be seen lies in the answer to the question, "Is it sufficiently likely that this drumhead is convex to warrant the administration of a general anæsthetic?" Personally I advise it in every case where a raised temperature and an ear-ache co-exist. It is then possible to remove the wax to clean the ear, and if necessary, to incise the drumhead under this. If the drumhead is in that state of fullness where one is uncertain whether an incision is needed or not, it is well to make it to save the possibility of another anæsthetic in a few hours' time.

When the drumhead, either by the art of the surgeon or in the course of Nature, is no longer intact, the duties of the practitioner are not ended. He still has two things to do: to attend to further treatment for the otitis media, and to watch the patient still for the first signs of mastoiditis. For the first day or so after the incision no further treatment is needed than to continue the instillation of the drops already advised, but on or about the third day it is well to mop the ear quite dry, and with the aid of the forehead mirror again to examine the drumhead with a view to seeing whether the free drainage is still present. This may not be free for two reasons. The healing power of the parts may have resulted in the incision having partly closed. This occurs occasionally, but not often if the incision has been free at the operation. More often it is the mere viscidity of the muco-pus which prevents its escape. In either case to

suck the middle ear empty of secretion and of pus is the best line of treatment. It is not in every child that this can be done, but with the earache relieved and the child used to the doctor it is surprising in how many cases it can be done. For this the old-fashioned vulcanite speculum of Siegel is more efficient than the "up-to-date" modification made of metal; it fits better, and does not cause nearly so much pain when gently pressed to plug the meatus. Often if the middle ear is once thoroughly emptied in this way it will heal altogether, otherwise the treatment should be repeated daily for three or four times. During this time the more often the ear is emptied of pus the better the chance of healing in the middle ear. This is best done by a wool plug on an appropriate carrier, but very gentle syringing with warm water will often do it quickest, especially if the discharge is thick and tenacious. Drops of hydrogen peroxide again are of value to bring the pus to the surface, where it can be more easily mopped away. When using these, however, it is important to remember that there is no virtue in them by themselves, and that except for their mechanical action they are valueless. This warning is necessary, because it would seem that there is a tendency to make a fetish of peroxide drops, as though to pour them in the ear were some libation which by itself would tend to make the middle ear well. Whichever method be used, it is necessary to dry the ear after; water is an irritant to the skin of the meatus, and this skin, like that in folds about the body, when inflamed needs to be kept dry. This is done by mopping, and the hygroscopic drops already mentioned complete the process.

When the ear has discharged itself the opening is much more likely to be inefficient, and this treatment should be instituted at once, but the hole in the drumhead may be so small that even with the negative pressure of the Siegel's speculum the secretion cannot be sucked out through it. Then an anæsthetic should be administered and the opening enlarged with the myrinogtomy knife. There should be no hesitation in doing this in every case in which there is the slightest doubt; if the pain does not disappear, if the temperature remains up, if the discharge is not free and there is evidence of its being pent up, operation should be done at once. To delay increases the risk of acute mastoiditis and of chronic suppuration. Of these the former is, if anything, the lesser of the two evils; it is usually promptly tackled, and there is many a person for whom in after life it would have been better had the original acute otitis media gone on to mastoiditis instead of being allowed to settle down into chronic suppuration. The appearance which most often suggests the need of this secondary incision is that of a red nipple-like process standing out from the drumhead and tipped with white. The appearance is quite distinct from that of a bulged drumhead; it is much more localised and

more conical than the oval lump of the bulge. At other times an efficient hole may be seen draining one part of the middle ear while a bulge in some other part indicates pus pent up locally. Here again an incision is needed in the part that still is bulging. After this secondary incision the same treatment should be carried out as after the primary one.

In this way the vast majority of cases of acute otitis media will be well in fourteen days, and some more will be well by the end of the month. The few remaining may be said to have passed over into the stage of chronic suppurative otitis media, and will have gone beyond the survey of this paper.

During all this time the practitioner must watch carefully for signs of mastoiditis. It is probable that in most cases of otitis media the mucous membrane of the antrum is inflamed ; when the inflammation has spread from the mucous membrane to the bone is a matter most difficult to say, and yet an attempt must be made to do so. It is not diagnosing mastoiditis to wait until a swelling appears behind the pinna : that is but dawdling till the fact is thrust upon you. There is no intention of going into this diagnosis here ; it is merely mentioned because to watch for mastoiditis is part of the treatment of otitis media. In a very doubtful case there is one form of treatment not yet mentioned. This is the old-fashioned one of the application of leeches. One may be put inside the pinna opposite to the posterior wall of the meatus and as near to it as possible, and another behind the ear over the cavity of the antrum. As with the other treatment, this depletion may just turn the scale in favour of recovery.

If the reader has followed the argument of this paper he will realise that the treatment recommended is just the same as that which is suited in other parts of the body to inflammation in a space surrounded by rigid walls, as, for instance, a whitlow of the finger ; the treatment may be summed up as being expectant until it is clear the condition will not subside, and then operative by simple incision without delay. The decision as to when the one should give place to the other is made by observation from day to day, or hour to hour. It is clear then that the consulting specialist is but seldom in the best position to apply this treatment. It is not often that he finds himself able to go and see the child from time to time. The treatment of acute otitis media in children is one that should be carried out in nearly every case by the general practitioner ; and to tackle the problem of chronic suppurative otitis media the treatment of acute otitis media needs far more attention paid to it. Till now the majority of men who qualify have had no opportunity of studying the subject. Within the last quarter of a century the general physicians and surgeons have handed over to the various specialists a large part of their

work, but have retained in their own hands an undue share of the teaching. To the few men who have taken an appointment in a special department these things have been taught, but there has been no opportunity for everyone to obtain this knowledge. The first step has been made to change this, and from now on every student must have studied in a department for diseases of the nose, throat and ear before he can go in for his final examination. It is to be regretted, however, that students will not always learn what they should in order to be useful practitioners in after life; they learn mainly or only those things about which they expect to be questioned in their "final." The next stage, then, must be that specialists be appointed to the examining bodies for the pass examinations for the finals. Then will the students expect to be asked questions in the essentials of otology, and then they will begin to learn it. This cannot happen in a day, but as the education of the profession improves so the treatment of acute otitis media will improve, and so the amount of chronic ear discharge be less.

Let it not be thought that this is the whole problem; perhaps it is but the lesser portion. It is clear that even to-day chronic discharge among the well-to-do is far less common than among the poor; it is doubtful if they get appreciably better treated. Try as hard as we can, our treatment is of small importance in comparison with the natural repair of the body. Among the poor, ill-fed in dirty homes, devoid of sunlight, the resistance is low compared with that among the children of the well-to-do. So we may conclude by saying that everything that is done to improve the conditions in which the poor live is a part of the treatment of acute ear disease in childhood.

THE NATURE OF BANTI'S DISEASE: ITS DIFFERENTIATION FROM OTHER TYPES OF SPLENOMEGALY AND ITS RELATION TO "IDIOPATHIC" NON-ALCOHOLIC PROGRESSIVE HEPATIC CIRRHOSIS IN CHILDREN AND YOUNG PERSONS.

By F. Parkes Weber, M.A., M.D., F.R.C.P.

The term *splenic anæmia*, whether in children or adults, has been generally applied to a chronic form of splenomegaly of unknown causation, accompanied by more or less persistent or intermittent anæmia, and tending to be followed by hepatic cirrhosis and ascites and often by jaundice.

It is for the stages of this disease or syndrome, in which the liver is involved, that the term *Banti's disease* has mostly been used. The question of diagnosis involves the recognition of many other varieties of chronic splenomegaly. The so-called *infantile form of splenic anæmia*—R. von Jaksch's *anæmia pseudoleukæmica infantum*—hardly comes into the question. It is occasionally associated with congenital syphilis, but perhaps it should be really looked on as an infantile type of *extreme hæmopoietic reaction* (doubtless essentially of conservative nature, from the modern vital teleological point of view) towards anæmia and grave nutritional disturbances produced by dietetic deficiencies, lack of sunlight and faulty general hygiene. This view would account for its association with rachitis, and also for its being somewhat less frequently met with now that the welfare of children of the poorer classes is being better understood and more attended to.

I will merely mention the splenomegalies due to *splenomegalic polycythæmia* (the Vaquez-Osler disease), *splenic tuberculosis*, the various forms of *leukæmia* and *aleukæmic lymphadenosis*, and the rare splenomegalies due to hydatid cysts and other *cystic conditions*.

Lymphogranulomatosis maligna (Hodgkin's disease) rarely commences with splenomegaly, but at various stages it may be of *splenic predominance*. I have discussed the diagnosis of this condition from other forms of chronic splenomegaly in a paper on " A Case of Late Hodgkin's Disease, with Remarks on Various Cases Presenting the Clinical Picture of Splenic Anæmia (Banti's Disease)" (1), and will here merely repeat, as I have remarked elsewhere, that *leucopenia*, to the significance of which too much reliance was formerly attached in the diagnosis of splenic anæmia and Banti's disease, may be met with in such cases as in *all kinds of chronic non-leukæmic splenomegaly*.

The possible confusion of *chronic septic splenomegaly* with splenic anæmia or Banti's disease was admirably illustrated by a case of chronic malignant endocarditis simulating splenic anæmia, which I described in 1910 (2), and which had been regarded as a case of splenic anæmia at several hospitals in London. Dr. Gordon Ward has quite recently dealt with the differential diagnosis of chronic septic splenomegaly (3).

There is a form of *chronic splenomegaly* not due to kala-azar and not rarely met with in *Egypt* and some other hot and tropical countries, which has been described by Day, Ferguson, Owen Richards (4) and others, and which in its main features is similar to Banti's disease. As the question of the diagnosis and treatment of such cases mainly concerns tropical medicine I need not enter into it here; neither need I discuss the cases of splenomegaly due to *chronic kala-azar* and *infantile Leishmaniosis* (as met with in Sicily and various parts of the Mediter-

ranean coast). Into the subject of *malarial splenomegaly* in children and adults I will likewise not enter.

The splenomegaly of *chronic acholuric* (*hæmolytic*) *jaundice* has certainly, especially before Chauffard's first paper on the subject, been confused with splenomegaly of splenic anæmia or Banti's disease. The cases of congenital hæmolytic jaundice in which there is some degree of splenomegaly but no definite jaundice present, may, as I have formerly elsewhere pointed out, be labelled *splenomegalic anæmia* (which they certainly are), to distinguish them from cases of splenic anæmia or Banti's disease. In cases of congenital hæmolytic jaundice it is especially in the intervals between the "attacks" or exacerbations of the disease that definite jaundice is sometimes absent. In congenital cases, even when there is more or less jaundice present (as there usually is), the patients often suffer so little that, as Chauffard said, they are "plus ictériques que malades." I am convinced that mild cases of the disease, when there is no decided jaundice and when the degree of splenomegaly is slight, often escape recognition in spite of all the numerous writings on the subject. In a family group of cases which I described with Dr. G. Dorner in 1910 (5) the condition in two of the cases (especially at a time when there was hardly any jaundice present) might have been easily overlooked. I am further strengthened in this opinion partly by having read a recent paper (6) on the subject, and partly by my colleague, Dr. E. Schwarz, having given me the opportunity of examining with him a girl, the subject of typical hæmolytic jaundice, and also her maternal uncle. We found that the sclerotics of the latter had a very slight icteric tinge, that his spleen was slightly enlarged, and that his urine showed a definite excess of urobilin and urobilinogen. He evidently, like his niece, was a subject of hæmolytic jaundice, but this had not been recognised by experienced physicians, who had previously been consulted by him for certain "attacks" or exacerbations, that were regarded at the time as attacks of cholelithiasis or cholecystitis. The other doctors had not, however, as we had, the advantage of knowing that his niece was a subject of typical hæmolytic jaundice. For us, under the circumstances, it was easy to make the diagnosis. Amongst familial cases there are certainly some that at an early stage of the disease can hardly be recognised in the absence of knowledge regarding other members of the family.

The Gaucher type of splenomegaly is very rare. I take the view, after F. S. Mandlebaum, that it is due to some metabolic disturbance and is not of the nature of a neoplastic formation. The "large clear cells," which are characteristic, if not absolutely pathognomonic, of "Gaucher's disease," have been found not only in the spleen, but in the liver, lymphatic glands and bone-marrow of subjects of the disease. Mandlebaum

concludes (7): "Fat or lipoid bodies cannot be found in the large cells by microchemical or polariscopic tests. The peculiar substance in the large cells does not lie in the extractive group, on chemical analysis, but is apparently of complex protein nature in combination with lipoids. The disease is evidently caused by some disturbance of metabolism, the products being found in a specific group of cells—reticulo-endothelial—of the hemopoietic system." I take it that the disease may be compared in some respects with cases of xanthomatosis and hæmochromatosis—but more especially with such congenital "inborn errors of metabolism" (in Sir A. E. Garrod's sense) as alkaptonuria, cystinuria, congenital hæmatoporphyrinuria, congenital steatorrhœa, and congenital functional abnormalities or deficiency of organs or tissues such as the bone-marrow (congenital hæmolytic jaundice and congenital hæmolytic anæmia and tendency to an aplastic type of anæmia). This view would amply account for the occasional familial occurrence of the Gaucher type of splenomegaly. The reticulo-endothelial system of cells is doubtless likewise largely concerned (but in another way) in the production of the symptoms of congenital hæmolytic jaundice—a disease which is recognised as frequently of familial occurrence.

Thrombotic splenomegalies have been sources of many errors in diagnosis, though the existence of chronic splenomegaly as a result of non-suppurative thrombosis of the splenic and portal veins (*adhesive pylephlebitis*) has been recognised for a long time. Quite a number of such cases have been clinically diagnosed as splenic anæmia or Banti's disease, even in the absence of marked anæmia; and in a few such cases the operation of splenectomy has been mistakenly undertaken. At the Clinical Section of the Royal Society of Medicine on January the 12th, 1912, I showed a case of "Chronic Splenomegaly of Uncertain Origin, with Persistent Leucopenia," and the sequel in 1915, for which I was indebted to Dr. H. M. Turnbull, of the London Hospital, showed that the case was one of non-suppurative thrombosis of the portal tract.(8) Many similar cases of progressive non-suppurative pylephlebitis, with periods of "clinical quiescence," have been reported, or are known of.

Syphilitic splenomegalies, especially those in children and young adults due to congenital syphilis, have frequently been regarded as splenomegalies of splenic anæmia or Banti's disease. Splenectomy has been successfully performed and has been followed by good results in some such cases. It has been suggested that in splenic anæmia the Wassermann reaction has been occasionally reported positive when syphilis was probably not present, and has wrongly led to the diagnosis of syphilitic splenomegaly. Anyhow, I believe that there is a group of splenomegaly cases in children in which hepatic cirrhosis is present or supervenes,

though often not recognised as cirrhosis for a long while—sometimes not till after death, at a post-mortem examination. The Wassermann reaction is not rarely positive in these cases, but I am not satisfied that in all these cases the disease of the spleen and liver is syphilitic in nature, though the positive Wassermann reaction may indicate that the disease is on a syphilitic basis.(9) Severe attacks of hæmatemesis may occur before the development of ascites.

Idiopathic Non-alcoholic Progressive Hepatic Cirrhosis in Children and Young Persons.

The question now arises: When all these various kinds of chronic splenomegaly have been excluded, are there any remaining cases that can be placed under the heading, splenic anæmia or Banti's disease? I believe there are, and I suggest that these remaining cases are varieties of a disease of unknown (so-called "idiopathic") origin, which I would call "idiopathic non-alcoholic progressive hepatic cirrhosis." This, as I take it, is a disease in which the liver is usually mainly affected, but in which splenomegaly may precede any sign of hepatic disease or both the spleen and the liver may early be enlarged. The "splenic" variety of this disease, in which the spleen is first and most obviously affected, I regard as being probably the same as "Banti's disease," if the latter term be restricted to the cases of chronic splenomegaly remaining after the exclusion of all the other above-mentioned forms of splenic enlargement.

Idiopathic non-alcoholic progressive hepatic cirrhosis, according to this view, is a disease which commences insidiously in childhood, but may not be recognised till much later in life, especially if the changes are almost confined to the liver. Ultimately, however, œdema of the ankles, ascites, and possibly attacks of hæmatemesis, jaundice and signs of hepatic insufficiency supervene. It is to the cases in which the disease is mainly hepatic in localisation that I think Dr. H. Batty Shaw (1922) referred in a paper on "Essential Cirrhosis of the Liver."(10) I agree with him in his suggestion that "alcoholic cirrhosis of the liver" is less often met with than formerly, and also that some of the cases formerly regarded as alcoholic cirrhosis of the liver may have really belonged to the non-alcoholic "essential" group.

Similarly, I would suggest that some of the cases in children, to which I have already alluded, of "Splenomegaly with Recurrent Jaundice, ending in Hepatic Cirrhosis and Ascites" (11), are really examples of the same disease, but with early splenic predominance—even when a positive Wassermann reaction is obtained; the positive reaction, however,

in such cases suggests that the splenic and hepatic changes may be " on a basis of congenital syphilis." In most cases both the spleen and the liver are early enlarged, in some cases the spleen more than the liver, in other cases the liver more than the spleen. The cases of Hanot's " biliary type " of hepatic cirrhosis in children and young persons, which are sometimes associated with great splenomegaly and are occasionally accompanied by general infantilism, may be examples of the same idiopathic hepatic cirrhosis, in which the cirrhotic change especially affects the minute bile-ducts. Very rarely more than one member of a family has been affected by hepatic cirrhosis in childhood, of Hanot's biliary type or of the ordinary type.(12)

If this view of non-alcoholic progressive hepatic cirrhosis, as I have explained it, be correct, the operation of splenectomy can of course be expected to be of use only in cases in which the morbid process is located mainly in the spleen (the cases of " splenic predominance ")—that is, in the cases of " splenic anæmia " or " Banti's disease," in which apparently it has repeatedly been found of great value.

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(12) In the ' Edinburgh Medical Journal' (1903, new series, xiv, p. 117) I referred to several examples of biliary hepatic cirrhosis occurring in two or more members of the same family. Byrom Bramwell (' Clinical Studies,' 1910, viii, p. 347) described a case of hepatic cirrhosis in a boy, aged 9 years. It was apparently of the ordinary type, but certainly not due to alcohol. Three other members of the same family had, according to the evidence, died from the same disease.

THE CLASSIFICATION OF BLOOD DISEASES IN CHILDHOOD.

By Gordon Ward, M.D.

In a recent series of articles in this Journal, Drs. Poynton, Thursfield and Paterson say: "It has appeared to us to be worth while to review and endeavour to classify as carefully as we are able the anæmias which have been met with in the wards of Great Ormond Street in the last two and a half years." I am distressed that these distinguished physicians were not bold enough to adventure further than the following classification (which includes only the actual series of cases with which they were dealing): (*a*) Congenital anæmias—acholuric jaundice, polycythæmia; (*b*) severe anæmias—anæmia gravid, von Jaksch's anæmia; (*c*) leukæmia; (*d*) simple anæmia—chlorotic type; (*e*) lymphadenoma; (*f*) purpura.

It is, of course, only too true that fools step in where angels fear to tread, but I must risk the imputation. Since I first published a detailed classification of blood diseases ten years ago the matter has been very frequently in my mind, and especially in relation to the many rare but curiously interesting syndromes published of recent years. My original classification dealt with forty different syndromes which might be brought under the head of "blood diseases," sometimes easily, sometimes with need for much explanation. The classification appended covers sixty syndromes, of which the great majority are met with in childhood. But the scheme of classification is unaltered, and these twenty additional syndromes fall easily into place beside their fellows. I venture to think that this fact is in some sort a justification of the original scheme.

The search for a first basis of classification required considerable thought. Ætiology proved quite useless. Syphilis can cause several different clinical pictures, *e. g.* Banti's disease, paroxysmal hæmoglobinuria and acquired hæmolytic jaundice. Tuberculosis can cause a simple anæmia by no means unlike that of chlorosis, or a generalised lymphatic involvement suggestive of lymphadenoma or leukæmia, or a chronic splenomegaly. Finally an anatomical basis was selected or, rather, a mixed anatomical and pathological. The main headings selected on these lines were:

(*a*) Generalised affections of blood-forming tissues, *i. e.* those diseases in which sufficiently thorough autopsies usually showed that the lesions were found in all the blood-forming organs.

(*b*) Localised affections of blood-forming tissues, *i. e.* those in which the disease appeared to pick out chiefly or exclusively a single one of the hæmopoietic tissues.

(*c*) Affections of red-cell formation and destruction, which really form a sub-group of localised affections.

(*d*) Affections of circulating red cells, *i. e.* those disorders in which the brunt of the disease is borne by the circulating cells.

(*e*) Affections of white-cell formation and destruction, to which the same remark applies.

(*f*) Affections of the circulating white cells, a group included for the sake of completeness rather than for any present importance which it has.

(*g*) Affections of the platelets, under which heading purpura must come in the present state of our knowledge, and in spite of the fact that we do not know whether the platelets are destroyed in purpura or fail to be formed in sufficient numbers.

(*h*) Affections of the plasma, including hæmophilia and chlorosis. It is no doubt true that cholæmia or hæmolytic jaundice and paroxysmal hæmoglobinuria might be brought into this group, and eventually it may be found to comprise nearly all the blood diseases. But that time is not yet.

Most of these groups were found to subdivide quite easily in one of three ways—on an ætiological basis, on an anatomico-pathological basis, or on a clinical basis. Groups *a*, *b*, *c*, *e*, and *h* have been first subdivided on an anatomico-pathological basis, groups *d* and *f* are divided on an ætiological basis into infective and toxic, and group *g* calls for no further subdivision.

These subdivisions increase the original 8 headings to a total of 22. Of these 22 those that seem to call for it have been further divided on a clinical basis into primary and secondary. These terms are not used in a very strict sense, but are meant to convey the meaning that the primary diseases are in general fully developed and often fatal syndromes of which the cause is not known, while the secondary are commonly much less complete and usually of known causation. The distinction is not absolute, but will, I think, be generally recognised as reflecting a useful clinical distinction between the two classes. The 22 divisions now become 33, including one instance in which "parasitic" and "septic" have replaced "primary" and "secondary." Of these 33 the largest group contains six different syndromes.

This classification necessarily assumes as reasonably probable the truth of various hypotheses to which general assent is not yet given. It may be that assent never will be given, and that the classification will have to be modified in the light of new facts. I should be interested to hear of any classification of which this could not be said.

In the meantime it stands as a plan for the co-ordination of existing knowledge which has served me well for some years, and which I venture to commend to the practical attention of my colleagues.

(a) **Generalised Affections of Blood-forming Tissues.**

1. Leukæmias :	
(*a*) Primary . . .	1. Lymphæmia
	2. Myelæmia
	3. Chloroma
(*b*) Secondary . . .	4. Infective lymphocytosis
	5. Myelæmias of cancer, sepsis, etc.
	6. Von Jaksch's disease
2. Hodgkin's disease . . .	7. Hodgkin's disease
3. Splenomegalies of Gaucher type	8. Gaucher splenomegaly
	9. Splenomegaly of Borrisowa, etc.
	10. Telangiectatic splenomegaly
4. Infective splenomegalies :	
(*a*) Parasitic . . .	11. Kala-azar
	12. Malaria
	13. Histoplasmosis
	14. Toxoplasmosis
	15. Krempf's splenomegaly
(*b*) Septic . . .	16. Syphilis, tuberculosis, etc.
	17. Non-specific, *e. g.* staphylococcal

(b) **Localised Affections of Blood-forming Tissues.**

1. Of the marrow :	
(*a*) Primary . . .	18. Multiple myeloma
(*b*) Secondary . . .	19. (Group C below)
2. Of lymphoid tissues :	
(*a*) Primary . . .	20. (?) Simple lymphoma
(*b*) Secondary . . .	21. Lymphatism
3. Of spleen	None

(c) **Affections of Red-cell Formation and Destruction.**

1. Increased formation :	
(*a*) Primary . . .	22. Erythræmia
(*b*) Secondary . . .	23. Secondary polycythæmia
	24. Blood crisis
	25. Post-hæmorrhagic anæmia
2. Decreased formation :	
(*a*) Primary . . .	26. Acute aplastic anæmia
	27. (?) Congenital anæmia

(*b*) Secondary . . .	28. Oligosideræmia
	29. Simple anæmia, septic, etc.
	30. X-ray and radium anæmias
	31. (?) Senile anæmia
3. Increased destruction :	
(*a*) Primary . . .	32. Congenital hæmolytic jaundice
	33. Dresbach's syndrome
	34. Herrick's anæmia
	35. Malins' syndrome
	36. Clough and Richter's syndrome
	37. Addisonian anæmia
(*b*) Secondary . . .	38. Acquired hæmolytic jaundice
	39. Intra-vascular cyto-phagocytosis
	40. Hæmolytic anæmias of Addisonian type, *e. g.* bothriocephaliasis
4. Disordered destruction . .	41. Hæmochromatosis
	42. Hæmatoporphyrinuria

(d) **Affections of Circulating Red Cells.**

1. Infective	43. Hæmoglobinuria
	44. Sulph-hæmoglobinæmia
	45. Infective methæmoglobinæmia
2. Toxic	46. Carbon monoxide poisoning
	47. Nitro-benzol, etc., poisoning (methæmoglobinæmia)
	48. Cyanogen poisoning

(e) **Affections of White-cell Formation and Destruction.**

1. Increased formation :	
(*a*) Primary . . .	(See generalised affections)
(*b*) Secondary . . .	49. Polymorph leucocytosis
	50. Eosinophil leucocytosis
	51. Mixed leucocytosis
	52. Lymphocytosis
2. Decreased formation :	
(*a*) Primary . . .	(In aplastic anæmias)
(*b*) Secondary . . .	53. Leucopenia
3. Increased destruction . .	(In Malins' syndrome)
4. Disordered destruction . .	(In Malins' syndrome)

(f) **Affections of Circulating White Cells.**		
1. Infective		(In Malins' syndrome)
2. Toxic		None
(g) **Affections of the Platelets.**		
1. (Mechanism unknown) . .		54. Purpura hæmorrhagica
		55. Chronic purpura
		56. Other purpuras
(h) **Affections of the Plasma.**		
1. Hæmophilias—(*a*) Primary .		57. Familial hæmophilia
(*b*) Secondary .		58. Hæmophilia neonatorum
		59. Hæmophilia calcipriva
2. Chlorosis— (*a*) Primary .		60. Chlorosis
(*b*) Secondary .		None.

A CASE OF CŒLIAC DISEASE SHOWING SYMPTOMS OF MEGALOCOLON, WITH AUTOPSY.

By Reginald Miller, M.D., F.R.C.P.,

Physician, Paddington Green Children's Hospital; Senior Physician to Out-Patients, St. Mary's Hospital, Paddington.

In recording the following case I have two purposes in view: First, I wish to establish that cœliac disease may give rise to such distension of the colon as to cause visible colonic peristalsis, and to suggest that some at least of the obscure examples of megalocolon which are met with in children past the age of infancy may be due to this malady. Secondly, I hope to support, so far as a single case is of use, the view which I have expressed elsewhere (6), that cœliac disease is a digestive fault and not an organic disease. The number of autopsies on undoubted cases of cœliac disease has been so very limited (3) that the record of even a single additional case is not without value.

Synopsis of Case.

The salient features of the case may be given very briefly. An undersized boy who died at the age of six years was under my observation for the last year of his life. When first seen he was admitted to hospital as an ordinary case of cœliac disease. His stools were typical, and on an ordinary diet contained 36 per cent. of fat. At that time it was specially noted that his colon was greatly distended, but that no peristalsis was visible. He was put on a modified low-fat diet and was sent to the Convalescent Home, where his diarrhœa continued.

Two months later he was readmitted, and for a period of six weeks very marked peristalsis of his colon was observed. The bowel appeared greatly distended : it held 75 oz. of saline without discomfort ensuing, and by skiagram an opaque enema showed that the sigmoid loop passed right across the lower abdomen to the right iliac fossa. He was treated by lavage of the colon.

Later he was admitted for a third time. The colon was then much distended, but no peristalsis was observed. His stools contained 60 per cent. of fat, dropping to 36 per cent. At this time he developed urinary obstruction due to a calculus impacted in his urethra, and thereafter showed infection of his urinary tract. Three months later he was re-admitted unconscious and dyspnœic, and died in a few hours.

Autopsy.—Omental fat was present in large quantities. The intestines appeared heavy, due partly to fat and partly (it was thought) to œdema. The colon was of normal size throughout, with the exception of one portion about $2\frac{1}{2}$ in. in length which was dilated to a width of a little more than 2 in. The presence of hypertrophy of the wall of the bowel, although suspected, could not be appreciated with certainty. No chronic enteritis nor other organic cause for the chronic fatty diarrhœa could be found. The gall-bladder contained coloured bile. Early pneumonia and chronic pyelo-cystitis were present.

Evidence connecting Cœliac Disease and Megalocolon.

Marked and persistent peristalsis of the colon without obvious cause is a phenomenon seen occasionally in children over the age of infancy. Such cases are uncommon, but not sufficiently rare to have failed to puzzle most of those interested in pædiatrics. They have never been satisfactorily connected with the " congenital " group of Hirschsprung. I suggest that this condition may be originated by the fat-indigestion of cœliac disease, in which disorder I think that the distension may reach such a grade as to produce partial obstruction by kinking with consequent peristalsis. In support of this view I submit the following evidence.

1. *Distension of colon in cœliac disease.*—Abdominal enlargement without signs of organic disease is one of the cardinal features of cœliac disease. Under the strictest diet and while free from symptoms cœliac cases show a prominent abdomen. With satisfactory progress the size of the abdomen does not so much become normal as the child " grows up" to the prominent abdomen. Under such circumstances the persistent enlargement is due to the deposit of fat in the omentum and abdominal wall combined with some laxity of the abdominal muscles, the result of the previous distension.

But in the stages where symptoms of fat-indigestion are present there is an additional cause for the abdominal enlargement, namely, flatulent distension of the colon. This is very noticeable in the cases with diarrhœa, but the essential connection between fat-indigestion and colonic distension is perhaps better brought out by study of those very mild cases which I have described as constituting the non-diarrhœic or " dry " type of the disease (5). In them, although no severe symptoms are present the colon can be seen enlarged, and there are complaints of distension, the passage of much flatus and occasionally discomfort or slight pain. Indeed, the connection between fat-indigestion and colonic distension becomes very clearly recognisable in such cases, so much so that the condition of the colon forms one of the best clinical tests as to the suitability of the amount of fat in the diet. Often this test is one of considerable delicacy, so that even a single meal in which too much fat is taken will be followed by visible distension and loss of appetite.

After the observation of a series of cases of cœliac disease, particularly those of the milder grades, I have no doubt that fat-indigestion can produce gaseous distension of the colon.

The size of the cœliac stool, weighing up to 1½ lb., and the decrease in the girth of the abdomen after an action of the bowels, are additional facts which suggest that an enlarged colon is commonly present in cœliac disease.

2. *Consideration of present case.*—In the case now reported we are able to trace the condition of the colon through sucessive stages. At first it was merely greatly distended; later, to the distension was added visible peristalsis. After a time this disappeared, and at the time of death the colon had practically regained its normal size.

The stage showing peristalsis lasted for some six weeks, and during that time the movements were very marked and appeared to be going on constantly. The colon was enormously dilated, the descending portion stretching right across to the right iliac fossa, in a position not unlikely to produce kinking. A colleague, who saw the child with me more than once, found no evidence of obstruction in the rectum, and the practically complete recovery of the bowel proves that the obstruction which produced the peristalsis must have been of a temporary nature. It is difficult to attribute it to anything else than a kink brought about by the excessive distension of the bowel by flatus. Microscopical examination of the colon showed what was thought to be hypertrophy of the muscle, but not to an absolutely definite degree.

3. *Consideration of Langmead's syndrome.*—In 1911, F. S. Langmead (2) described under the title of " Colonic Tetany " a very interesting group of cases which showed a symptom-complex characterised by relapsing

tetany, dilatation of the colon, abnormal, offensive motions, and in most instances retarded growth and development. In several of these cases visible colonic peristalsis was present.

The relationship between the more chronic of Langmead's cases and cœliac disease is hardly disputable. At the time of publication the author stated his cases closely resembled those described by Herter (1) under the name of "intestinal infantilism." It is now generally agreed that Herter's cases were really examples of Gee's cœliac disease, and re-perusing the details of Langmead's cases it is clear, in the light of knowledge which has accrued since the publication of his paper, that they are, in many instances at least, examples of cœliac disease of a type sufficiently severe to be called cœliac infantilism. This, perhaps, explains why no further examples of the fully developed syndrome have been reported, though many instances of cœliac disease associated with tetany have been published. In my present case neither tetany nor facial irritability was found at any time.

It is interesting to note that Langmead found that out of 10 cases of fatal tetany, renal calculi were present in three instances. It seems likely to be more than a mere coincidence that in my present case a stone was passed *per urethram*.

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ON LOCALISED PERITONITIS DUE TO INTESTINAL PERFORATION IN PARATYPHOID FEVER: REPORT OF A CASE.

By A. E. Mortimer Woolf, F.R.C.S.,

Surgeon to Queen Mary's Hospital for East End, etc.

Perforation in paratyphoid fever is uncommon. Webb-Johnson (7), reviewing 2500 cases of typhoid and paratyphoid fevers, found that out of 1038 cases of paratyphoid B, only 3 cases (0·29 per cent.) of perforation occurred. Perforation was more frequent in the paratyphoid A cases, viz. 2 out of 344 (0·85 per cent). The percentage of perforation in paratyphoid A and B together amounted to 0·36 per cent. On the other

hand, Torrens and Whittington in 1915 estimated the frequency of perforation in paratyphoid fever at 1 per cent. The variation in these two estimates may be due to the fact that the former series was described in 1917, and that nearly one-third of the cases had been inoculated against either paratyphoid A or paratyphoid B. According to Vincent (6) and Muratet, whose very complete work on typhoid and paratyphoid fevers has been translated by Dr. J. D. Rolleston, the infrequency of perforation in paratyphoid fever is true only in civil practice; during the war hæmorrhage and perforation appeared almost as frequently in paratyphoid as in typhoid fever. The various statistics they quote in support of their statement referred chiefly to a small series of cases.

A study of the literature has revealed that when the intestine perforates in the course of a typhoid or a paratyphoid fever, general peritonitis nearly always ensues. Two cases of paratyphoid appendicitis quoted by Lord Dawson and Dr. Whittington (1) also gave rise to general peritonitis, with death. One of these cases was complicated by severe and deep ulceration for 5 feet above the ileo-cæcal valve; the appendix was gangrenous. In their second case a perforation due to a paratyphoid ulcer in the proximal third of the appendix had occurred. These cases are noteworthy, as it might be imagined that if localised peritonitis had occurred owing to perforation it would be more likely to happen when the appendix was the seat of the perforation.

Webb-Johnson (7), in his book, mentions that perforation may be protected by local plastic peritonitis. He refers to two cases of typhoid in which an attempt had been made by the omentum to localise the effects of perforation. He also draws attention to the fact that bands may subsequently be formed, and give rise to intestinal obstruction.

The following case is so rare and interesting that it seems worthy of record:

Paratyphoid B; perforation; formation of localised abscess; operation; recovery.—The patient was a boy, aged 15 years, undersized and of poor general physique. He had suffered from tuberculosis of the cervical glands. There was a scar of an old incision on the right side of the neck.

On August the 26th, 1921, he was seized with fever and headache, and was first seen by a doctor two days later, on August the 28th. While at home he was not dieted for enteric fever, and suffered from constipation and not diarrhœa. On September the 8th he was removed to the Chadwell Heath Isolation Hospital, with a diagnosis of enteric fever.

On admission the boy had a normal temperature, pulse and respiration. He was constipated, the tongue slightly furred, abdomen doughy, but not distended, no tenderness nor rigidity—the spleen was just palpable at the costal margin. Examination of the blood by microscopical Widal reaction showed partial agglutination of *B. typhosus*, and complete agglutination of *B. paratyphosus* B in dilution of 1 in 50 in thirty minutes.

On the afternoon of September the 9th the boy complained of some discomfort in the abdomen, and in the early morning of the 10th, of abdominal pain. At 11 a.m., when seen by Dr. Burton (Medical Officer of Health), there was acute pain over the right side of the abdomen, with tenderness in the right loin and right iliac region. When seen by

me at about 4 p.m. the boy looked ill, and the whole of the right side of the abdomen was rigid and tender. A mass, situated high up and far out on the right side of the abdomen, could be felt. The mass was tender, it did not move on respiration, and could not be pushed back into the loin to be felt bimanually. The temperature had risen to 100° F., pulse 104.

Immediate operation was performed. The mass was found to consist of the great omentum, which had rotated to the right, and was obviously delimiting an inflammatory focus. The appearance was similar to the walling-off of an appendicular abscess by the great omentum, especially when that abscess is situated above the right iliac fossa. The general peritoneal cavity was packed off and the omentum was gently separated. The perforation of the intestine immediately appeared, through which a large quantity of white material escaped. At this point the intestine and cæcum were so matted that it was impossible to say which part of the intestine was perforated, but judging by the escape of this white material, I believe that the lower end of the ileum was affected. The perforation was closed by two sutures, but owing to the great œdema of the intestinal wall, it was impossible to insert a reinforcing suture layer. Every time a stitch was tightened it immediately cut out. The omentum was therefore brought over the site of the perforation and sewn round. A tube was inserted.

Recovery was uneventful.*

I have been able to find recorded in the literature a few cases of abscess formation due to typhoid fever, but I can find no examples recorded in cases of either paratyphoid A or B.

As early as 1873, Murchison (5) described three cases of localised abscess which occurred during the course of enteric fever, all of which recovered, although no operation was performed. Two of these patients evacuated their abscesses *per rectum*, one after two months, the other after " several months." In the third case the abscess burst externally after 6 months.

On the other hand not all of these cases have such a happy termination, even though the attainment of it be prolonged. Perforation may occur in more than one place. This is especially liable to occur when protective adhesions do not occur, and is one of the causes of the high mortality, even if operation be carried out early. A sequence of perforations may, however, occur when protective peritoneal adhesions are abundantly present, as in a case reported by Gautier (2) and Brutsch of the Geneva Medical Clinic.

The patient was a man, aged 26 years. The onset of the typhoid attack was mild, the patient doing his work for fifteen days. Perforation then suddenly occurred, the peritonitis rapidly becoming localised. From time to time the symptoms became more severe and then subsided. At autopsy an adhesive peritonitis was found localised in right iliac fossa, and also a sac containing fæcal matter. The intestine showed multiple perforations, including one in the large intestine, and the authors consider that there is little doubt that the exacerbation of symptoms which occurred from time to time was due to a fresh perforation.

Gioseffi (3) has also recorded a case in a boy, aged 6 years, in whom a typhoid ulcer in a Meckel's diverticulum was found post-mortem to have

* The patient was shown six months later at a meeting of the Chelsea Clinical Society.

given rise to a localised adhesive peritonitis. The child also had endocarditis and was too ill on admission to allow of operation.

Keen (4) quotes a case of his own, in which a localised abscess was formed and a fæcal fistula resulted. Six months later he operated, and after a very troublesome dissection, the fistula was found to end in a somewhat globular mass under the abdominal wall. The opening into the intestine could not be discovered. The mass was curetted, disinfected and packed. The fistula contracted after the operation so that two or three days elapsed without discharge. The patient died, however, from general debility nine weeks after her discharge from hospital.

There can be little doubt that the ideal treatment of these cases is early evacuation of the abscess and closure of the perforation. As in the case reported above, the closure of the perforation may not be altogether easy to accomplish. The ideal method is to place one's sutures as near the margin of the perforation as possible, and to reinforce these by either one or two layers of Lembert sutures placed on each side, so that when tied the sutured margins of the perforations are invaginated. Unfortunately the walls of the intestine are so infiltrated that the tying of the reinforcing sutures makes them cut out. The same difficulty occurs when a purse-string suture is placed around the closed perforation. From the analogy of perforated gastric and duodenal ulcers, in which there is also much infiltration, either of the stomach or of the duodenal wall, purse-string sutures when tied are even more likely to cut out than a series of Lembert sutures. Usually it is not possible to place these reinforcing sutures, and too much time should not be wasted in attempting to achieve the impossible. Further damage to the intestinal wall will be the only result. The best method is to bring over a piece of the great omentum and to fasten it by a few sutures around the closed perforation. This acts even more efficiently than either one or two reinforcing layers, and has the great advantage of simulating Nature's method of cure. Moreover, this method involves little or no tension on the infiltrated intestinal wall.

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THE MENINGEAL FORM OF TYPHUS IN CHILDREN.*

By MM. Gratzosky, A. Ströe and E. Cocias.

Typhus in children occasionally assumes the meningeal form which one of us (A. S.) described in adults during the war. In the Children's Clinic at Bucharest we recently had occasion to observe three cases of this kind. The first was a child aged 2 years admitted at the onset of the disease with a high temperature, bloated features, congested conjunctivæ, and a very distinct meningeal syndrome, consisting of strabismus, inequality of the pupils, nuchal rigidity, exaggerated reflexes, ankle clonus, and positive Kernig and Brudzinski signs. On lumbar puncture a clear fluid was obtained under hypertension, in which a thick and friable film formed in twenty-four hours. The cytological formula was as follows : a large quantity of lymphocytes (15–20), a few red corpuscles and basophil cells, and scanty polymorphonuclears. Nonne and Boveri reactions positive. Urea 0·324 per mille. No micro-organisms. Weil-Felix reaction positive in the blood in 1 : 200 ; Widal's reaction for typhoid, paratyphoid A and B negative. Death took place in coma. The necropsy showed meningeal infiltration and a large quantity of cerebrospinal fluid, slightly blood-stained in character. Vessels of pia mater dilated up to their finest ramifications, giving the brain a red tint. Small hæmorrhagic meningeal suffusions. Pink colour of grey substance with the vessels gaping on section. Increase of fluid in the ventricles—in a word, the typical lesions of typhus. Examination of the other organs showed myocarditis, fatty degeneration of the liver, and a slight degree of nephritis.

The other two cases, one a child aged 3 years and the other a child aged 7 years, showed the same appearance on admission. One child also had a slight eruption. The Weil-Felix reaction in the blood and cerebrospinal fluid was the same as in the first case. Both recovered in nine and ten days respectively. The conclusions to be drawn from these cases help in the differential diagnosis of typhus from meningitis. The symptoms as well as the character of the cerebrospinal fluid should be carefully studied. The abortive course of typhus in these cases is characteristic. The cerebrospinal fluid in typhus, as Slatineanu-Galasescu, Daniélopolu, Devaux and others have shown, possesses special features, consisting in abundant lymphocytosis and occasional presence of red corpuscles and polymorphonuclears. The meningeal states described by Widal are differentiated from the meningeal form of typhus by the absence of the Weil-Felix reaction.

* A paper read before the Société médicale des Hôpitaux de Bucarest on October the 11th, 1922.

Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, October the 27th, 1922.

The President, Dr. Eric Pritchard, *in the Chair.*

The Pathogenesis of Rickets.—Dr. Eric Pritchard pointed out in his presidential address that most of the known ætiological factors of rickets were merely contributory causes, and that no one of them was the *causa vera*. He believed that the true cause was the production in the system of an excessive quantity of acid bodies other than CO_2 which necessitated neutralisation at the expense of the alkali reserves of the body. Although ammonium, sodium, potassium and iron were bases which were requisitioned for this purpose, calcium itself was not exempt from this toll. The main clinical and pathological evidence of rickets was calcium depletion, and the main symptoms those to which calcium depletion of bone, muscle and nerve gave rise. He further pointed out that all the recognised ætiological factors of rickets contributed to the general stock of acid bodies poured into the blood, by interfering in some way with the complete oxidation of the food products consumed. If food elements were not completely oxidised, acid bodies other than CO_2 and of larger molecular size were necessarily produced. All depressants of metabolism or oxidation such as want of sunshine, want of exercise, bad housing, heat, air stagnation, etc., were also contributory factors in the production of rickets; while all stimulants of metabolism such as sunlight, cold air, moving air, exercise, and psychological conditions such as joy, pleasure, play, ambition, curiosity, were also therapeutic expedients in the cure. Everything which damaged the animal body as a working machine tended to the production of incomplete products of oxidation, and there might be many combinations of such contributory factors.

Rickets was a composite disease consisting of the various symptoms due to the component contributory conditions, and of the special symptoms due to the acid bodies produced in excess (acidosis), and to the general mineral depletion. The address was illustrated by diagrams which helped to explain the general argument.—(*Author's abstract.*)

Two Cases of Duodenal Obstruction in Infants.—Dr. R. C. Jewesbury, after alluding to Dr. E. Cautley's paper on duodenal stenosis in the British Journal of Children's Diseases, 1919, xvi, p. 65, reported the following two cases:—Case 1: Female child. History of vomiting during last five months of pregnancy. The child born before this died at nine days with a history of vomiting and constipation since birth. Present illness: Full-term child, apparently normal. Vomiting began on third day and continued two or three times a day about one hour after feeds, except on the 13th, 14th and 15th days. Vomiting projectile. The vomit was bile-stained. Child never cried; slept all the time. Bottle-fed with diluted cow's milk. Nineteen days old when admitted to hospital; very wasted; weight 4 lb. 9½ oz.; lay with legs drawn up. After admission vomited after

every feed. Abdomen poorly covered, marked peristalsis from left to right. A rounded swelling extended from the left costal margin downwards below the umbilicus up to the right costal margin, where a small indefinite mass was felt. Peristalsis occurred every half to one minute. X-ray report: Dilatation of the first and second part of duodenum and greatly distended stomach. Very little food had left in two hours. Probably duodenal obstruction.

The child passed small infrequent motions and continued to vomit until death, which occurred on the 29th day. Autopsy: Œsophagus normal. Stomach grossly hypertrophied, pylorus not thickened and pyloric lumen larger than normal. First and second parts of duodenum dilated and walls thickened owing to obstruction at third part. Between the second and third parts of the duodenum was a much constricted portion about $\frac{1}{4}$ in. long which admitted an ordinary probe. There was no evidence of obstruction from surrounding structures.

This appeared to have been a case of true congenital stenosis of the duodenum.

Case 2: Male infant, aged 9 days. Family history: Parents healthy; one other child delicate. No miscarriage. Full-term child, breast-fed; bowels had been acting daily.

Present illness: Vomiting, projectile in character, began on third day and had persisted since. Vomit was bile-stained. The child was rather wasted and slightly jaundiced; the stomach was dilated and showed peristalsis from left to right. The stools were intermediate in character between meconium and milky stools. After giving 2 oz. of breast-milk by the bottle the child was in evident discomfort, and almost immediately vomited with great force; the vomit was bile-stained and was more in amount than the small quantity taken. Vomiting occurred after every feed. X rays showed that the stomach retained most of meal for nineteen hours. The appearance suggested constriction of first and second parts of duodenum. The condition remained the same until death on the thirteenth day. Autopsy: The stomach and duodenum were enormously dilated and hypertrophied. Below the duodeno-jejunal junction the bowel was very shrunken and small. The cæcum was situated abnormally high under the liver, and the obstruction in the duodenum was caused by pressure from outside by surrounding structures and in particular by the right colic artery. The obstruction therefore was not due primarily to any abnormal narrowing of the duodenum itself, but to pressure on it from surrounding structures.

Friday, November the 24th, 1922.

Case of Cerebral Degeneration.—Dr. Helen Ingleby showed a girl who was born in April, 1920. She had a fall on her head at birth, but recovered and progressed normally until she was 20 months old, when she had an attack of broncho-pneumonia and whooping-cough. Towards the end of January, 1922, about a month after the attack of broncho-pneumonia, she began to fall about, her muscles wasted considerably, and swallowing became so difficult that she had to be fed by bottle. Epileptiform fits started at Easter and became very frequent. There was incontinence of urine and fæces. Lumbar puncture showed a great increase of pressure in the cerebro-spinal fluid, which contained a trace of albumin but no excess of cells or organisms. The Wassermann reaction was negative in the blood

and cerebro-spinal fluid. Dr. Ingleby suggested that the condition was a cerebral degeneration akin to amaurotic family idiocy.

Double Tumour in the Perinæal Region of a Child.—Dr. Bernard Myers showed a child aged 3½ months with two small swellings in the perinæum, one about the size of a pigeon's egg and the other a little smaller. They were situated ¾ in. in front of the anus and to the left of the middle line. The tumours were sessile, of smooth surface and covered with skin. Their consistency suggested that they were lipomata.

Case of Persistent Jaundice in an Infant; Atresia of the Common Bile-duct and Biliary Cirrhosis.—Dr. Bernard Myers.—The infant, which died at the age of 14 weeks, had had persistent jaundice since 2 days after birth. The motions were white, and the urine distinctly bile-coloured. Icterus neonatorum had been present in the mother's 3 other children, disappearing in 4 or 5 days, but there was no similar case in the family. The autopsy showed atresia of the common bile-duct and biliary cirrhosis.

Case for Diagnosis.—Dr. Bernard Myers showed a child, aged 3½ months, with the following condition: Both elbow-joints were extended, forearms pronated, wrists and fingers flexed, the thumbs being inserted under the fingers towards the bases of the little fingers. The limbs were rigidly held in this position slightly in front of the body. Attempts to place the thumb in a normal position evidently caused pain and the child cried at once. The knee-joints were extended and the toes pointed, the heels being drawn up. A decided degree of flexion was present at the hip-joints, the lower extremities being thus placed towards the front of the body. It was very difficult to overcome the spasms of the ankle-joints and toes. The Wassermann reaction was negative. Mentally the child was bright and alert, and the cranial nerves appeared to act normally. The muscles round the shoulder and pelvic girdles were very small and apparently weak. All the muscles of the upper arm and thigh more particularly and those of the forearm and leg to a lesser degree showed a certain amount of atrophy. The muscles of the back seemed to be less affected. No changes in the discs or fundi. Cerebro-spinal fluid normal.

Case for Diagnosis? Renal Dwarfism.—Dr. G. M. Kendall showed a girl, aged 4½ years, who had been apparently healthy up to the age of 2½, when she had an acute illness and was sent to hospital. On admission she presented general œdema and ascites which appeared and disappeared during her 4½ months' stay. There were also diarrhœa and vomiting, which recurred at intervals. Urine contained no albumin. Wassermann reaction positive in patient and the mother, negative in the father. Patient was treated with mercury and a course of N.A.B. intravenous injections were given. On readmission to hospital 2 years later, in addition to œdema and gastro-intestinal disturbance, she presented well-marked genu valgum. The urine at first showed no albumin, but later it contained a faint trace. The fundi were normal. Blood-pressure 80/55. The Wassermann reaction was now negative. X rays showed that the ends of the diaphyses of the tibiæ and femora were translucent and striated longitudinally; the epiphysial line was straight. The long bones of the upper limbs were also translucent, but more irregular; there was not the same longitudinal striation and the

epiphysial line was not straight. There was periosteal thickening of the shafts. The evidence of renal disease was not very conclusive, but the X-ray appearances were very like those published by Barber in 'Quart. Journ. Med.' April, 1921. Only 1 of the previous cases on record had a positive Wassermann reaction. The patient was only $1\frac{1}{2}$ in. below the average height. In most of the other cases the dwarfing was very marked, but the deformity had appeared later in life and the patients were older when first seen.

Cœliac Infantilism in the Convalescent Stage.—Dr. R. Miller showed two cases of cœliac infantilism in the non-diarrhœic stage to demonstrate (1) that at this stage the stools still showed an excessive fat wastage, and (2) that if the excess of fæcal fat was chiefly in the form of soaps, the stool might be dark, even constipated, and far from fatty in appearance. Dr. Miller pointed out that there were three forms of fatty stool: First, the type of pancreatic insufficiency, with a large excess of fat in the stools, chiefly in the form of neutral fat. This condition never arose in cœliac disease. Secondly, that of excess of fat chiefly in the form of fatty acids, giving the classical cœliac stool, large, pale, unformed and very offensive. Thirdly, there was the soapy stool, looking normal on casual inspection; it was not offensive and not frequent, yet it might contain up to 50 per cent. of fat, chiefly in the form of soaps. This stool was found in the non-diarrhœic stages of the disease. The only obvious alteration in it from the normal was its increased bulk.

In treatment an attempt should be made (1) to converts fats into soaps, (2) to increase the fat absorption.

Hereditary Tylosis.—Dr. J. D. Rolleston (*v.* British Journal of Children's Diseases, 1923, xx, p. 16).

Rickets Treated by Light Therapy.—Dr. R. C. Jewesbury showed a negro child, aged $2\frac{1}{2}$ years, who had been treated by daily exposure of the whole body to the radiations from a carbon arc lamp. Skiagrams were taken at fortnightly intervals; the last, which was taken 6 weeks after the first, showed that the rickets was almost healed.

Pneumoccocal Septicæmia and Enlargement of the Liver and Spleen.—Dr. R. C. Jewesbury showed a girl, aged $3\frac{1}{2}$ years, who had been admitted to hospital with 2 days' history of abdominal pain. The abdomen was tender and somewhat distended; the liver and spleen were very considerably enlarged, the spleen in proportion being larger than the liver. A moderate degree of ascites was present. The temperature was 104°F. on admission and there had since been irregular pyrexia. Blood examination: Red cells, 3 million hæmoglobin 55 per cent., leucocytes over 12,000. Differential count showed nothing particular. Blood culture showed a pure growth of pneumococcus. Wassermann reaction negative.

Inborn Errors of Metabolism.—Dr. Donald Paterson showed three cases. (1) Congenital steatorrhœa. The boy, aged 3 years, was brought to hospital with the complaint that he was passing fat in the stools. He was one of nine children, all well. The parents appeared to be healthy. He had had no previous illnesses, but it was noticed that when he was weaned the motions contained fat. Occasionally he passed several stools in the day, but

usually only one, and it was never formed. The motion shown looked like thick yellow vaseline and had a strong smell like that of butter or cheese. Analysis showed that more than 99 per cent. was composed of fat, of which only 8 per cent. was split. (2) Two cases of congenital cystinuria. Two children in the same family, aged 4 and 2 years respectively, continually passed cystin in the urine. The elder had a calculus removed from the bladder when he was 2 years old, and the younger had had a calculus removed quite recently. Both calculi were composed of cystin. An attempt was being made to control the formation of cystin by a low protein diet, and to prevent the cystin from being precipitated by giving sodium bicarbonate to keep the urine alkaline.

CLINICAL SECTION.

December the 8th, 1922.

Case of Acromegaly in a Girl with Congenital Aortic Stenosis.—Dr. E. Stolkind.—The patient was a girl, aged 16 years. Congenital heart disease was diagnosed in infancy. At the age of 6 she had scarlet fever and at 7 measles. From 5 to 13 she suffered from nocturnal enuresis. Menstruation began at 15, at first every two months, and later every month for 2 days and in slight quantity. She showed all the signs of aortic stenosis. Apex beat and cardiac dulness were about half an inch to the left of the nipple line. There was a systolic thrill, and a rough loud systolic murmur in the aorta (less audible at the apex) conducted to the carotids. The second aortic sound was slightly accentuated. X-ray examination showed that the heart was slightly enlarged downwards and to the left with a rounded apex.

The patient left school at the age of 14 as a backward girl. Growth had been fast during the last 7 years, and very rapid during the last 2 years. She was now 5 ft. 5·9 in. high and weighed 9 st. 7½ lb. The length of her feet was 10¾ in. and of the middle fingers 4⅖ in. She was taller than her parents and elder sister, aged 19 years. There was some polyphagia, but no polydipsia or polyuria. No albumin or sugar in urine. The patient was very weak and soon became tired after exertion. She often dropped things which she held in her hand. She was very shaky and had been stooping for some months past. She had no memory, was mentally slow and took no interest in anything. The superciliary areas were abnormally developed. The lower jaw was visibly prognathic.

X-ray examination showed that the posterior clinoid processes, which were rather large for a girl of her age, ended in a fragment of bone hanging backwards. The sella turcica was large, but within normal limits. The nasal sinuses were large, and there were slight signs of acromegalic changes in the supra-orbital ridge and mandible. The X-ray signs were those of acromegalic gigantism, which had not yet reached the stage of sellar malformation. The eyes were normal. Treatment consisted in X-ray applications. Dr. Stolkind, who referred to his paper on hereditary syphilitic aortitis (*vide* British Journal of Children's Diseases, 1920, xvii, p. 126), remarked that a combination of acromegaly in a girl, aged 16 years, —juvenile acromegaly—with congenital aortic stenosis was very rare.

SECTION OF DERMATOLOGY.

December the 21st, 1922.

Case of Bullous Eruption.—Dr. J. H. Sequeira.—The patient was a boy, aged 3½ years. Family history unimportant. The child's general health was good. There was no evidence of visceral disease, but for the last 6 months there had been an eruption of bullæ, which came out singly and in groups, varying in size from that of a split-pea to that of half a cob-nut. They contained perfectly clear serum and the base at the onset was free from erythema. Practically the whole surface was covered with groups of blebs. There was some irritation at night, but the child took its food well. The temperature was quite normal. There was slight eosinophilia of the blood (10 per cent.), but no eosinophils in the serum. At first the blebs were free from organisms, but later developed *Staphylococcus epidermidis albus.* The question whether the case was pemphigus or dermatitis herpetiformis was purely one of nomenclature. If the term "pemphigus" meant a bullous eruption where the blebs came out on a clear skin and where the disease was not essentially polymorphous, the case should be classed as pemphigus. From his past experience Dr. Sequeira thought that the condition would react well to arsenic.

Case of Onychatrophia.—Dr. E. G. Graham Little showed a girl, aged 10 years, who began to develop changes in the nails at the age of 5. One by one the affected fingers had lost their nails; apparently they had been absorbed, very much as in epidermolysis bullosa, but there was no other evidence of that disease. The toes were quite normal.

In the subsequent discussion, Dr. H. G. Adamson (President) said he had seen a similar nail condition in acquired syphilis. The child might have congenital syphilis, and he suggested that a Wassermann reaction should be performed.

Dr. F. Parkes Weber suggested that the case was allied to Raynaud's disease.

January the 18th, 1923.

Case of Keloid after Burns.—Dr. E. G. Graham Little showed a boy, aged 11 years, who had received a burn 8 months ago, and now showed a keloidal growth on the site of the burn covering almost all the middle surface of the thigh. An interesting feature was that part of the large patch had undergone spontaneous healing. Parts of the tumour-formation were spreading in the typical crab-like way, while other parts had undergone spontaneous healing. Dr. Graham Little proposed to have the main area treated with radium, because it was very itchy and painful, and the boy's rest was badly disturbed by it.

Light Baths in Tuberculous Disease of the Skin.—Dr. J. H. Sequeira showed two boys suffering from a grave type of lupus who had derived considerable benefit from light baths. A strong carbon-arc lamp was fixed 3 feet from the ground and 8 patients at a time, wearing only bathing drawers and screened from the draught, sat in a circle round the

lamp, exposing back and front alternately, the eyes being guarded by thick veils. A patient was first put under the influence of the lamp for half-an-hour, the dosage being gradually increased up to four hours a day.

SECTION OF LARYNGOLOGY.

February the 2nd, 1923.

Simple Fibrous Strictures of the Œsophagus.—Mr. A. Ryland.—The patient was a child, aged 3 years, who in August, 1922, swallowed caustic soda, which was followed by increased difficulty in swallowing. On November the 6th skiagrams were taken and treatment commenced. At this time the child was only able to take liquid nourishment slowly. By œsophagoscopy the upper stricture was seen and located at 14 or 15 cm. from the upper incisor teeth. The stricture was seen to be small and annular in character and the lumen was displaced posteriorly. No cicatricial bands in sockets were visible. Dilatation was commenced by means of No. 1 gum-elastic urethral catheter. On December the 6th No. 12 catheter was passed without difficulty through the upper stricture to a distance of 18 cm. from the upper incisor teeth.

The child was now able to take soft solids, *e. g.* bread and butter, etc. On January the 11th No. 12 hollow bougie filled with barium sulphate was seen on the screen to be arrested abruptly at a distance of 18 cm. from the upper incisors. No. 5 bougie entered the stomach fairly easily. The third stricture, which was situated at 21 cm. from the upper incisor teeth, was the narrowest and most intractable.

Mr. Ryland had had some success by making repeated efforts at dilatation with graduated bougies, and proposed to persevere with the treatment at frequent intervals.

SECTION OF OTOLOGY.

December the 15th, 1922.

Otitis Media with Facial Palsy following Scarlet Fever.—Mr. F. J. Cleminson.—The patient was a girl, aged 2 years and 10 months, who had been brought to hospital on September the 8th, 1922, for a discharge from both ears. There was pus in the left meatus and incomplete right facial palsy. The history given by the mother was that the child was well and had normal hearing up to April the 25th, 1922, when she was admitted to an isolation hospital for scarlet fever. While there she developed measles and nasal diphtheria. She was discharged on September the 1st. The patient was sent to have her right ear syringed by a nurse, who brought back a perfectly macerated malleus and incus without any signs of caries. The nurse had seen the ossicles evacuated with the lotion and had rescued them from it. The opposite ear being in a state of chronic purulent inflammation had been subjected to a radical mastoid operation and had become dry. The patient was apparently totally deaf. While under observation the condition of the lower part of the face (especially that of the muscles retracting the angle of the mouth) had improved very noticeably.

Parotid Fistula following Mastoid Operations. — Mr. Norman Patterson.—The patient was a girl, aged 12 years. History of right otorrhœa on and off since infancy. When seen on September the 19th, 1921, there was a history of severe pain, purulent discharge from right ear, redness and bulging posterior meatal wall; posterior perforation; tenderness over mastoid. Temperature 100·5° F., pulse 120; respirations 24. Operation same day. Complete mastoidectomy. Bone cellular, cholesteatoma in middle ear; lateral sinus exposed and appeared healthy. September the 21st, temperature 103°, pulse 120. Wound opened up. Decomposing blood-clots and sloughs removed. Lateral sinus appeared healthy. On November the 26th the temperature, which had fallen to normal, rose to 102°. Under anæsthetic more sloughs were removed, and the lateral sinus was found to contain a parietal clot. The operation was completed by ligaturing the internal jugular vein. Examination of cerebro-spinal fluid obtained by lumbar puncture negative. Hæmolytic streptococcus given from blood-culture. For a week swinging temperature up to 104°. The temperature then came slowly down to normal, and the patient was sent to a convalescent hospital on November the 28th. On February the 1st, 1922, a swelling was noticed in the operation scar. The swelling burst; the discharge was at first blood-stained, then clear. On May the 22nd the wound was opened up and curetted; it was discovered that the sinuses did not lead into the mastoid cavity. Present condition: middle ear healed. Depressed scar over mastoid with very small opening, from which saliva persistently drained. The flow was increased by chewing. Diastase was present in the fluid in large amount.

February the 16th, 1923.

Necrosis of Temporal Bone following Scarlet Fever, Measles and Diphtheria.—Mr. J. F. O'Malley showed a child aged 7 years who had been admitted to hospital with concurrent scarlet fever and measles. A fortnight later diphtheria supervened, followed by necrosis of the left temporal bone involving the facial nerve and labyrinth. Dead bone was removed and a plastic operation performed. Present state: wound healed; occasional slight otorrhœa; facial paralysis less apparent. Vestibular test: cold caloric, no response; C_2 tuning-fork (right ear closed) not heard.

Société de Pédiatrie, Paris.

June the 20th, 1922.

Treatment of Acute Articular Rheumatism by the Intravenous Injection of Salicylate of Soda.—MM. Guinon and Hirschberg, commenting on M. Lesné's communication at the last meeting (see British Journal of Children's Diseases, 1923, xx, p. 41), stated that they had employed intravenous injection of salicylate of soda in 10 cases of acute articular rheumatism in children, but without obtaining any conclusive results as regards relief of pain, reduction of temperature or prevention of cardiac lesions. On the other hand certain disagreeable effects occurred, viz. violent delirium in one case and bradycardia in another.

Treatment of Nocturnal Incontinence by Acidification of the Urine.—M. Carrière, of Lille, referring to M. Zuber's paper (*v.* British Journal of Children's Diseases, 1923, xx, p. 41), stated that since 1902 he had emphasised the importance of testing the reaction of the urine in cases of enuresis, and had been in the habit of giving the acid phosphate of soda if the urine was alkaline. Cases of enuresis, however, with alkaline urine were not very frequent, as he had found this factor present in only 16 per cent. out of 400 cases of enuresis seen in the course of twenty-five years.

Adiposo-genital Syndrome; Malformation of the Four Extremities; Agenesis of Two Incisors; Hereditary Syphilis.—MM. E. Apert and Broca.—The patient was a boy, aged 9 years. In the right hand the thumb and little finger were normal, but the other 3 fingers were atrophied and grouped together. The left hand was almost normal apart from incurvation of the index. The toes were very small and the 3 central ones were webbed. The lower 2 central incisors were absent. The Wassermann reaction was positive in the mother and negative in the child.

Infantile Dystrophy with Mental Defect and Heredo-syphilitic Meningitis.—MM. P. Lereboullet and E. Pichon.—The patient was a girl, aged 13½ years. The dystrophy consisted in defective mental and physical growth and nasal and dental malformation, and the meningitis was shown by considerable spinal lymphocytosis, inequality of the pupils, and loss of the pupillary reflexes to light and accommodation. The Wassermann reaction was positive in the blood and cerebro-spinal fluid.

Hypothyroidism, Pulmonary Stenosis, Psychical Disturbance and Chorio-retinitis.—MM. P. Merklen and M. Minvielle.—The patient was a boy, aged 16 years. All the lesions were congenital and due to inherited syphilis. Desmoulière's reaction was completely positive in the patient, negative in his sister, who was healthy, and much attenuated in the mother, in whom the disease was practically extinct.

Tuberculous Splenomegaly—MM. Nobécourt and Nadal reported this case in a boy, aged 7 years, which presented the following features of interest. Clinically the diagnosis could be made only by exclusion. Syphilis might have been suspected owing to familial strabismus and cranial rickets, but the Wassermann reaction was negative in the patient, his mother and two sisters. The absence of parasites in the blood excluded malaria and kala-azar. There was no new growth to suggest involvement of the spleen. Absence of changes in the blood excluded leukæmia and pseudo-leukæmic anæmia. On the other hand the swinging temperature and presence of tracheo-bronchial adenopathy indicated tuberculosis as the cause. Pathologically, apart from miliary granulations in various organs, there were only two old caseified tubercles in the mediastinum and a small tubercle in the left lung. The spleen, on the other hand, showed large tuberculous lesions, consisting of two caseous tubercles each the size of a Mandarin orange. The splenic tissue remained only at the periphery. It was therefore a case of primary tuberculosis of the spleen—a very rare condition in children, of which Lefas in the Paris thesis of 1903 could find only three cases on record.

Acute Primary Pneumococcal Peritonitis in Infants.—MM. Ribadeau-Dumas and J. Meyer remarked that most cases of pneumococcal peritonitis in infants were secondary to pneumococcal infection of the lungs. They reported two fatal cases of primary pneumococcal peritonitis in infants, aged 6 weeks and 10 weeks respectively, in which a correct diagnosis was not made until just before death, which took place without operation after a few days' illness. The cases showed that a sudden onset, vomiting and bilious diarrhœa, especially in breast-fed children, an anxious expression, and later the formation of an umbilical fistula in the absence of any previous local suppuration, were indications of primary peritonitis. Although the prognosis in such cases was very grave, it might be improved by early diagnosis and surgical drainage, accompanied by repeated intra-peritoneal and subcutaneous injections of large doses of anti-pneumococcal serum.

A Fresh Case of Pyloric Stenosis cured without Operation.—MM. Pehu and J. Rhenter, of Lyons.—The patient was the fifth child of a family in which the eldest had died of repeated vomiting at the age of 3 months. The three other children were normal, except one who had vomited frequently during the first year of life. The patient started vomiting three weeks after birth, and showed the same intolerance for breast milk, sterilised milk and whey. On examination of the abdomen peristaltic waves were frequently observed, and contraction of the stomach was felt. The stools were scanty. Loss of weight and cachexia resulted. Improvement and finally recovery took place after washing out the stomach at first twice a day for eight days, and then once a day for a fortnight.

Cystic Sarcoma Simulating Pleuro-peritoneal Tuberculosis.—MM. Armand-Delille, Isaac-Georges and Ducrohet.—The patient was a girl, aged 13 years, who died suddenly with symptoms of severe dyspnœa after two months' illness in which the progressive enlargement of the abdomen, pain in the chest, dry cough and fever suggested tuberculosis of the peritoneum and pleura. The autopsy showed a tumour the size of an adult's hand adherent to the appendix, pelvic colon and small intestines, and arising from the left adnexa. Microscopical examination showed it to be a round-celled sarcoma which had undergone cystic degeneration.

In the subsequent discussion M. Veau referred to a similar case which simulated peritoneal tuberculosis, but where that diagnosis was excluded owing to a negative cuti-reaction, and a large mucoid cyst of the ovary was found on laparotomy. Recovery took place.

Lumbar Puncture in Chorea.—M. Taillens, of Lausanne, recorded eight cases of chorea in children, aged from 8 to 12½ years, in whom he had employed lumbar puncture. His conclusions were as follows: (1) Hypertension though frequent, was not the rule in chorea; (2) lymphocytosis was not found; (3) the sequelæ which occurred in 2 cases appeared 2–4 days after puncture, and consisted in headache and vomiting; (4) the therapeutic effect was *nil*.

Pseudo-tetanic Eclampsia in Pertussis.—M. T. Reh, of Geneva, recorded a fatal case in a female infant, aged 20 months, of pertussis complicated by convulsions simulating those of tetanus. The autopsy showed

no evidence of encephalitis, but merely hyperæmia of the meninges and parathyroids with broncho-pneumonia.

Prophylaxis of Diphtheria in the Infant.—MM. Lesné, Boutillier and Langeron examined Schick's reaction in 226 cases aged from 1 month to 20 years and over, and found that the percentage of negative cases was about the same in the infant under one year and in the adult. In the artificially fed infant immunity to diphtheria disappeared by the end of the first year. The breast-fed child, on the other hand, remained immune for a longer period. Inherited immunity, however, was only temporary. A negative reaction might become positive any day—a fact which explained the occurrence of diphtheria in children whose reaction was negative (Blechmann, Chevalley, Blauner). The test, therefore, was of no help in the prophylaxis of diphtheria in the infant. As diphtheria is likely to run a severe course in infants under one year, and as they did not readily acquire immunity from anti-diphtheritic vaccination before the age of 18 months, injection of serum was the only effective method of prophylaxis at this age.

Meningeal Symptoms in a Child of Syphilitic Parents, probably due to Cerebral Tumour.—MM. L. Guinon and F. Hirschberg recorded the case of a boy, aged 10 years, admitted to hospital with meningeal symptoms. The cerebro-spinal fluid was under pressure, clear, and showed distinct lymphocytosis. Some improvement took place under treatment with arsenobenzol and mercury, but the symptoms recurred. Examination of the fundus showed papillary stasis. After decompression trephining the headache disappeared and the fundus became practically normal.

Bromide Eruption of the Papulo-vegetative Type.—MM. L. Guinon and Hirschberg also showed a female infant, aged 8 months, who, as the result of a draught containing about 0·20 grm. daily given by the mother for restlessness, developed large papillomatous lesions on the lower limbs, especially on the left thigh, where she had been vaccinated. The appearance of the lesions was absolutely typical and sufficient to exclude syphilis or tubercle. On cessation of the bromide mixture they rapidly faded, but resumed their typical appearance in two days on resumption of the drug.

Suppurative Aseptic Arthritis of the Shoulder-joint in an Infant with Parrot's Pseudo-paralysis; Recovery.—M. G. L. Hallez reported a case of syphilitic osteochondritis of the upper epiphysis of the left shoulder in a child, aged 4 weeks, with non-microbial suppurative arthritis and periarthritis of purely syphilitic nature. Complete recovery took place under mercurial treatment, showing that the condition was not so grave as Parrot supposed.

Gangrenous Purpura in Scarlet Fever during Serum Sickness.—MM. Lesné, Lefèvre and Laffitte reported the case of a boy, aged $3\frac{1}{2}$ years, admitted to hospital with scarlet fever and membrane on the tonsils from which diphtheria bacilli were cultivated. 30 c.c. of diphtheria antitoxin were injected subcutaneously and 20 c.c. intramuscularly on admission, and 20 c.c. subcutaneously the following day. Eleven days

later a generalised severe urticaria developed, fresh crops appearing for a week. Seven days after the first appearance of the rash purpuric spots appeared on the right calf and right buttock and increased in size during the next few days, in which similar lesions appeared on the elbows, right pinna and left calf. All the lesions presented the same dark blue colour with rounded and definite outline except on the buttock, where they were surrounded by a diffuse mottling. The lesions and neighbouring region were anæsthetic. Death took place four days after appearance of the first purpuric spot. A blood culture was negative. Examination of the blood showed: red cells, 3,040,000; leucocytes, 31,000; polymorphs, 58·8 per cent.; large mononuclears, 9 per cent.; medium sized, 8 per cent.; hæmoglobin, 55 per cent. Post mortem a large infarct was found in the right kidney and thymus. The suprarenals were normal.

October the 17th, 1922.

Congenital Hemi-hypertrophy. — MM. Babonneix and Buizard alluded to the cases recently reported by Black-Milne (*v.* British Journal of Children's Diseases, 1920, xvii, p. 79), and recorded a case in a boy, aged 4½ years, of congenital hemi-hypertrophy localised on the right side, and accompanied by (1) extensive nævi on the left lower limb, left part of the scrotum, anterior part of the penis and occiput. (2) A transverse vein at the root of the nose without any other evidence of heredo-syphilis apart from slight hydrocele. The Wassermann reaction was negative both in the mother and child. (3) Absence of hyperplasia of the right testis. X-ray examination did not show any gross changes in the skeleton apart from a slight increase in size of the bones of the limbs on the right side. As in one of Black-Milne's cases, the possibility of an hereditary influence was shown by a paternal uncle presenting a similar malformation.

Coxa Vara; Transplantation of the Lower End of the Gluteus Medius on to the Shaft of the Femur.—MM. Veau and Lamy.—The result of this operation, which was performed on a boy at the age of 14⅕ years, was most successful, the patient being able to walk, run and jump two years after the operation. The coxa vara had been slight on the right side and very marked on the left. The operation had apparently been performed only once before.

Infantile Eczema caused by Prolonged Consumption of Woman's Milk containing a considerable Excess of Butter.—MM. Marfan and Turquety (*v.* British Journal of Children's Diseases, 1923, xx, p. 53).

A Case of Chorea Cured by Lumbar Puncture.—M. Cassoute of Marseilles.—The patient was a girl, aged 9 years, in whom 10 c.c. of cerebro-spinal fluid were withdrawn under hypertension. Before lumbar puncture the choreic movements were very distinct, but the following day they had disappeared and did not return during the month which the patient remained in hospital, although she was not given any drug treatment.

A Case of Tetanus Cured by Large Doses of Serum.—MM. L. Tixier and H. Duval reported a case of severe tetanus which developed after an incubation period of 5 days in a boy, aged 11½ years, who had contracted a

contused wound of the left thenar eminence by a fall on a brick. 40 c.c. of tetanus antitoxin were injected for 10 consecutive days, half subcutaneously and half intramuscularly, and then 2 c.c. for 4 days, or a total of 408 c.c. in 14 days. Subcutaneous injections of carbolic acid were also given in doses of 1 c.c. of a 3 per cent. solution for 10 consecutive days. Complete recovery took place without any serum reaction.

A Case of Tetanus Cured by Large Doses of Serum.—MM. Nobécourt, Nadal and R. Mathieu also reported a case in a boy, aged 8 years, who developed tetanus 7 days after a wound on the knee. 400 c.c. of antitoxin were injected, the greater part (310 c.c.) within the first 5 days. 270 c.c. were injected intramuscularly. Two injections (50 and 20 c.c.) were given intraspinally during the first two days. The last injection (60 c.c.) was given subcutaneously.

In the subsequent discussion M. Comby referred to four cases which he had published some years ago (*v.* British Journal of Children's Diseases, 1915, xii, p. 1), successfully treated by subcutaneous injections of tetanus antitoxin.

Tuberculous Infection of the Newborn.—MM. R. Ribadeux-Dumas, J. Meyer and Demerliac recorded the case of a male infant born on July the 6th, at the seventh month, who presented a positive cuti-reaction one month after birth (August the 8th), and died on September the 22nd of pulmonary tuberculosis, characterised by a primary focus of lobular pneumonia at the right base and secondary miliary tuberculosis in both lungs. The mother, an old tubercular subject, had developed galloping phthisis after delivery, but nevertheless suckled the child for 20 days.

On the Importance of Exact Measurements in Therapeutics, and Especially in Infantile Diabetics.—M. G. Railliet, of Rheims, emphasised the importance of having tablespoons, dessertspoons and teaspoons containing 15 grm. and 5 grm. respectively manufactured under the control of the service of weights and measures.

Two Cases of Habitual Vomiting Cured by Mercury.—M. H. Dorlencourt alluded to the recent communication of MM. Marfan and Lemaire on the frequency of congenital syphilis in infants suffering from habitual vomiting (*v.* British Journal of Children's Diseases, 1922, xix, p. 39), and recorded two cases of habitual vomiting in which all the ordinary methods of treatment had failed, but recovery almost immediately took place as the result of anti-syphilitic treatment (lactate of mercury). Neither infant showed any other clinical evidence of syphilis, but the Wassermann reaction was positive in the only one in which it was performed.

Infantile Cholera with Symptoms of Acidosis, Treated by Intraperitoneal Injections of Bicarbonate of Soda followed by Intolerance for Milk.—MM. Ribadeaux-Dumas and J. Meyer.—The patient was a male infant, who at the age of 5 months developed fever, vomiting and diarrhœa, associated with symptoms of acidosis, consisting in sighing respiration, peculiar odour of the breath and absolute anuria. After intraperitoneal injection of 50 c.c. of a 5 per cent. solution of sodium bicarbonate marked improvement took place and the symptoms of acidosis disappeared, but for a considerable time the child showed an intolerance for milk.

Rheumatoid Purpura; Death from Intussusception.—MM. L. Tixier and Canouet recorded this case in a boy, aged 11 years. Death took place without operation. The autopsy showed an intussusception about 30 c.c. in length in the middle of the small intestine with a small perforation, which had given rise to an encysted peritonitis.

Syndrome of the Occipito-cerebello-vertebral Angle; Tubercle of Cerebellum; Operation; Recovery.—MM. P. Nobécourt and Paraf.—The patient was a boy, aged 9½ years, who presented the following symptoms: (1) Paresis of the external branch of the spinal accessory, manifested by a latero-posterior inclination of the head with a small atrophic furrow and muscular twitching; (2) anæsthesia in the region of the second occipital nerve; (3) slight cerebellar signs, consisting in titubation, vertigo and adiadodokinesis. An exploratory operation was performed, and a tubercle the size of a hazel-nut was found in the cerebellum. Rapid recovery took place after its removal.

Barlow's Disease in an Infant fed on Fresh Milk.—M. H. Lemaire recorded a case in a female infant, aged 17 months, who presented all the symptoms of Barlow's disease after being fed with a milk which had been pasteurised, but had not been treated with hydrogen peroxide or any other preservative. The mother stated that she had boiled it only once before putting it in the bottle.

In the subsequent discussion M. Marfan remarked that milk which had been pasteurised and subsequently boiled might give rise to scurvy. Neumann, of Berlin, had recorded cases similar to that of M. Lemaire. Individual predisposition was also an important factor.

A Case of Non-congenital Myotonia.—M. H. Lemaire recorded the case of a male infant who was absolutely normal up to the age of 6 months, when he contracted a short febrile disease, in which myotonia gradually developed, and reached its height at the age of 13 months.

The myotonia, which was first shown by the child being unable to hold its head up or sit up, subsequently spread to the limbs. There was no reaction of degeneration, but merely a diminution of contraction to the faradic current. At the age of 3½ years the myotonia began to disappear, so that by 4 the child had recovered a good deal of his muscular power and was able to stand and walk, and the electrical reactions were normal.

Abstracts from Current Literature.

Alimentary Diseases.

The building of good teeth and their preservation (*'Journ. State Med.,'* 1922, xxx, *p.* 480).—**H. Scurfield** points out that the importance of sound teeth to good health is being more and more recognised. He puts forward a programme for the prevention of dental decay as follows: (1) A diet containing a good supply of vitamins for all, but especially for expectant and nursing mothers, weanlings and adolescents. Such a diet we

can get by using plenty of milk, animal fat, eggs, green vegetables and fruit, cod-liver oil being very valuable as a stand-by. (2) Attention to the teeth of expectant and nursing mothers, as this has a noteworthy effect on their ability to breast-feed. (3) For the weanling food that compels mastication so that its jaw muscles and tongue may have exercise. (4) Avoidance of chronic catarrh or any other obstruction which would prevent the infant from breathing through its nose. (5) Measures to keep the teeth clear of sugar or starch at all times of the day. To give a child milk and biscuits after it has gone to bed is bad. Each meal should be ended with a tooth-cleansing food, such as apple, orange, celery, lettuce, nuts, crisp toast, etc. Sweets, if eaten, should be taken during the meals, and sweets, jam, honey or milk should not be the last food taken at a meal. A child which has been taught to masticate will not want to wash down half masticated food with water. A drink at the end of a meal may help to clean the mouth, but the flow of saliva and the natural movements of the mouth should be sufficient in well-formed jaws if the meal is not ended with a sticky food. The acid of fruit stimulates the salivary flow and helps the natural flushing. The object is to keep the teeth clear of fermentable sugar and starch between meals, and some may like to trust to the use of the tooth-brush after a meal. J. ALLAN.

The beginnings of disease stopped by the building and preservation of good teeth (*'Journ. State Med.,'* 1922, xxx, *p.* 486).—**S. Barwise** urges that education and maternity and child welfare committees should see: (1) That the prospective mother is suitably fed on foods rich in vitamins; (2) that the infant is breast-fed; (3) that in the second year of life it is not fed on food containing excessive carbohydrate equivalent and that vitamins are supplied to make up the deficiency caused by the sterilisation of the milk consumed; (4) that after the age of two the child is fed on solid food and is taught only to drink after meals; (5) that from two onwards the child is fed on such foods as will make it use its jaws, so strengthening and broadening them, and is given hard foods to scrape and preserve the teeth. J. ALLAN.

Physiological prevention of dental caries and oral sepsis (*'Journ. State Med.,'* 1922, xxx, *p.* 484).—**J. Wheatley** looks upon dental caries as a most significant sign-post indicating departure from physiological living in one very important respect. The following simple rules have been drawn up and acted upon with the object of getting good development of jaws and regular teeth and keeping the teeth from decaying. Sufficient food requiring vigorous mastication should be given with every meal. This should begin in an infant 8 or 9 months old and be gradually increased, so that at 4 or 5 years a large proportion of the food should be given in a form needing thorough mastication. No liquid should be taken at meal times, but between meals, and preferably half an hour to an hour before meals. Except for children under 3 years of age three meals a day should be the rule, and no food, particularly sweets, should be taken between meals. A meal should be finished with a cleansing food, *e. g.* with fruit, uncooked vegetables, or, in the absence of these, with a tough crust or some other substance requiring vigorous mastication. Finishing a meal with jam, marmalade or milk is especially to be avoided. Milk and biscuits, chocolates or sweets the last thing at night are particularly dangerous and these foods should never be taken at this time. J. ALLAN.

Some causes of irregular teeth and their effects (*'Journ. State Med.,'* 1922, xxx, *p.* 489).—**A. E. Barnett** states that irregular teeth are the expression of imperfect bone development, the bones concerned being the jaws, which are subservient to and designed for the support of the teeth, All factors interfering with the growth and development of the jaws must be remedied, *e. g.* improper feeding, nasal obstruction, bad habits, such as thumb-sucking, lip-sucking, etc. As to the evil effects of irregular teeth there is lack of function resulting in a lessened ability to masticate the food properly. There is increased liability to disease such as caries and pyorrhœa alveolaris, and there is a predisposition to mouth-breathing, with a decreased resistance to disease in general and a peculiar susceptibility to affections of the ear, nose and throat. J. Allan.

The teeth in syphilis and tuberculosis (*'La Pediatria,'* 1922, xxx, *p.* 823).—**C. D'Alise** examined 206 children at the dental clinic at Naples and found them suffering from 21 varieties of dental anomaly; 92, or 44 per cent., were syphilitic, 119, or 58 per cent., were tuberculous, 59, or 29 per cent., had both diseases, 24, or 11·6 per cent., were healthy, and 30, or 14 per cent., had indefinite complaints. He found typical Hutchinson teeth only in 2 cases. Carabelli's tubercle was met with not only in the first upper permanent molar, but often also in the second upper milk molar and was not connected with hereditary syphilis. Microdontism belonged almost always to the first dentition and was very frequent, but usually limited to the incisors and canines and was not associated with syphilis only, but was also frequent in tuberculosis. There was a constant relation between the various forms of malocclusion and tuberculosis. Vincent Dickinson.

Congenital hypertrophy of the pylorus (*'Brit. Med. Journ.,'* 1923, i, *p.* 579).—**G. F. Still** considers 248 cases in detail; 210 of these were boys and 37 girls. About half of them were firstborn children. In 4 instances 2 cases of this affection had occurred in the same family. The commonest date of onset is the third week. Vomiting, constipation and wasting are the three outstanding symptoms. The vomiting of indigestion is usually associated with diarrhœa and with abnormal stools. The latter are normal in congenital hypertrophy of the pylorus. The vomiting is of insidious origin and gradually gets worse. The vomit consists of food only in the beginning, but as time goes on much glairy mucus is added. Bile is sometimes present, proving that the closure of the pylorus is not continuous. The loss of weight is usually 5–6 oz. a week. Each change of food is apt to be followed by lessening or cessation of the vomiting for 24–48 hours. The only absolute proof of the existence of pyloric hypertrophy is the presence of a tumour varying in thickness from that of a lead pencil to that of a hazel nut. It is situated 1 to 1½ in. above the level of the umbilicus close to the right side of the vertebral column, thus necessitating deep pressure under the right rectus muscle. The tumour disappears as the pylorus relaxes. Only in 2 cases of the 248 was there failure to palpate the tumour. Palpation may have to be continued for 10–15 minutes, and the infant should be fed during the examination, thereby ensuring relaxation of the abdomen, the promotion of peristalsis, and the displacement of the pylorus to the right. 225 cases are considered as regards the success or failure of treatment; 156 recovered and 69 died. Far better results were obtained in private cases. Of 127 in this class 113 recovered,

while of 98 hospital cases only 43 survived. This is partly due to the fact that wet-nurses are obtainable for private but not for hospital cases. Again, in hospital infants are very prone to develop gastric enteritis. Six of the 156 recoveries were treated by simple dietetic methods. Lavage was tried in 78 cases; 43 recovered and 35 died. This procedure must be continued for 10–16 weeks. It should be abandoned if the weight continues to fall, even for a few days. Increase of weight is usually not recorded for 7–10 days. A great advantage of operation over lavage is that breast-feeding can be continued. 29 cases were submitted to Rammstedt's operation, *i.e.* longitudinal incision through the peritoneal coat and muscular layers of the pylorus, leaving the mucosa intact; 18 of the 29 recovered and 11 died from collapse, shock or hæmorrhage, the direct result of the operation. Forcible dilatation was performed in 108; 86 of these recovered and 22 died; 80 were undertaken in private with only 6 deaths, 28 in hospital with only 12 recoveries. 16 deaths were due, not so much to the operation, as to gastric enteritis and marasmus. Recurrence was only noted in 3 of the cases treated by forcible dilatation and in 1 of the Rammstedt series. CHRISTOPHER ROLLESTON.

Congenital hypertrophy of the pylorus (*'Edin. Med. Journ.,'* 1921, I, *p.* 1).—**J. Thomson** records his personal experience in 100 consecutive cases treated during the last twenty-five years. The subjects specially dealt with comprise the family history and complications, the causation of the muscular hypertrophy and of the symptoms, the natural course of the disease under medical and surgical treatment, the symptoms and choice of treatment in different types of the disease, the diagnosis, especially that between pyloric hypertrophy and the so-called "pyloric spasm," the difference in the prognosis in hospital and private cases, and the subsequent health of the patients who recover. A table of the cases is appended. J. ALLAN.

Pyloric stenosis from muscular hypertrophy in the infant (*'Gaz. des Hôp.,'* 1921, XCIV, *pp.* 1221 and 1253).—**H. Godard** says this affection is rare in France but frequent among the Anglo-Saxon races, and is much the most common in boys. The symptoms generally begin abruptly in the fourth week, with violent vomiting, constipation and rapid emaciation. The vomiting is explosive, and the ejected matter is large in quantity and never contains bile. Peristaltic waves over the stomach region are a very important physical sign, and often the enlarged pylorus may be palpated. After some weeks when gastric dilatation has taken place the vomiting becomes less frequent but larger in the amount brought up, and containing milk clots (indicating gastric stasis) and having a smell of butyric acid, and sometimes being frothy. The writer distinguishes four forms: (1) the acute with severe symptoms; (2) chronic; (3) a slow form developing during childhood; (4) an abortive form which by many is considered a spasm of the pylorus. The X-ray plate, showing that a bismuth meal remains in the stomach at the end of 4 hours, makes the diagnosis certain and surgical interference imperative; if 80 per cent. of the bismuth mixture has passed in 4 hours the case may be treated medically. The diagnosis may have to be made from gastro-enteritis, cardiospasm, vomiting due to gastro-neurosis, congenital stenosis of the pylorus or duodenum, or pylorospasm. Slight forms are often cured by careful diet and the administration of an alkali and an antispasmodic such as opium or belladonna. In severe forms operation is necessary, and the author recommends the pylorotomy designed

by Fredet, but often known as Rammstedt's operation, which has the advantage of ease, rapidity, and absence of shock.

J. Porter Parkinson.

Congenital pyloric stenosis (*'Lancet,'* 1922, i, *p.* 944).—**R. Eccles Smith** states that stenosis is caused by antenatal spasm due to hyperadrenalism reinforced by (1) inhibition of pancreatic secretion; (2) superadded gastritis; (3) spasm of pylorus initiated by phimosis; (4) spasm due to unknown causes. The case reviewed occurred in a male infant of 3 weeks. Vomiting occurred after the first feed. There were emaciation and constipation. Rammstedt's operation was performed, but the accompanying phimosis was not treated. Progress was satisfactory till the sixteenth day after operation when vomiting recurred. Circumcision was performed, resulting in cure. At the age of 6 months a right inguinal swelling was noticed but not operated on till 18 months later, when a large appendix was found in the sac. The author considers that an attack of acute appendicitis or a pull of a fixed and adherent appendix on the duodeno-jejunal junction might have set up an increased activity of the pyloric sphincter.

Christopher Rolleston.

Hyperchlorhydria in childhood (*'New York Med. Journ.,'* 1921, cxiv, *p.* 226).—**L. Berman** describes this as occurring between the first and second dentition, with the following symptoms: impaired appetite, abdominal pain, nausea and vomiting. Gastric analysis shows an excess of hydrochloric acid. The abdominal pain is variable, sometimes directly after meals, or in the morning or night, or on waking the child from sleep. The children affected are usually dark complexioned, with low blood-pressure, low blood-sugar and relatively increased fatigability.

J. Porter Parkinson.

Carcinoma of the stomach (*'Edin. Med. Journ.,'* 1921, i, *p.* 98).—**D. A. Laird** reports a case in a boy, aged 15 years, who had complained of pain of a fortnight's duration, with occasional vomiting. There was no suggestive hereditary or personal medical history. On examination there was a palpable epigastric tumour. The liver was enlarged and there was leucocytosis. There was no X-ray evidence of value. Progressive and rapid loss of weight occurred. On laparotomy being performed an inoperable growth was found. Temporary improvement followed, but subsequently the disease progressed and terminated fatally. Microscopic examination showed a columnar-celled carcinoma of the lesser curvature of the stomach with extensive metastases to lymphatic glands—gastric, pyloric, aortic mediastinal—to the liver, the pancreas and the diaphragm. J. Allan.

Hirschsprung's disease (*'Arch. Ped.,'* 1923, xl, *p.* 49).—**M. Reuben, L. Silver** and **W. Steffen** state that this disease was described by Hirschsprung in 1886, but before this 25 cases had been described, the first by Billard in 1820. The essential elements of this malady are dilatation and hypertrophy of the whole colon, especially the sigmoid, without obstruction, and its existence from birth. It is far more common in boys than girls, and is very rarely hereditary. Out of 70 cases, 41, or nearly 60 per cent., were under 4 years of age, which points to the fatal character of the complaint. The authors consider that the excessive length of the sigmoid causes kinking, which produces partial or complete obstruction.

The chief symptom is constipation, which may be present from birth. A history of not having the bowels open for a week is common, and periods of 5, 6, 9 and 12 weeks are recorded. Diarrhœa may alternate with constipation. The amount of abdominal distension varies with the severity of constipation, is greatly reduced by diarrhœa, but never disappears entirely. Peristalsis is visible but seldom painful. Secondary symptoms due to toxins are malnutrition, anæmia, fatigue and dyspnœa. On palpation a tumour is not infrequently felt, which in reality consists of fæcal masses. On percussion there is a general tympany, any dull areas being due to fæcal impaction. On auscultation borborygmi may be heard. The sphincter ani is normal. The stools during constipation are dry and inspissated. At other times they are like putty and of a yellowish, brownish or greenish colour. They are very offensive. The large intestine is the site of the pathological change and in one-third of the cases the sigmoid is alone involved. The distended coils are sometimes 6–8 in. in diameter. The serous coat is roughened and there may be peritonitis. Microscopically all layers, especially the muscular, are hypertrophied. Very few cases go on to adult life. Death may be due to colitis, ileus, perforation, peritonitis, or pulmonary intercurrent infections. Surgical is more effective than medical treatment. Meat, chicken and fish should be avoided, and replaced by eggs, milk and carbohydrates. CHRISTOPHER ROLLESTON.

Two cases of congenital megacolon (*'La Pediatria,'* 1921, XXIX, *p.* 209).—**V. Provinciali** describes these cases in boys of 9 and 13 months; in the former, which resulted in recovery, radiography showed evidence of a mechanical and functional stenosis of the sigmoid, in the latter death ensued from follicular colitis. The author strongly supports the idea of a congenital malformation as opposed to that of a fœtal infection.

VINCENT DICKINSON.

The intestinal flora in infants (*'New York State Journ. Med.,'* 1922, XXII, *p.* 155).—**H. Sherman** and **H. R. Lohnes** state that bacterial end-products are either harmless acids, or are split proteid products, which are often toxic in nature. The infantile flora differ from the adult flora in that the type has not become fixed. Thus it can readily adapt itself to a change of food. Through improper feeding the type may become either excessively fermentative or excessively proteolytic. These types can be differentiated by culture media and are often associated with clinical symptoms.

J. ALLAN.

"Colic" in the nursing infant (*'New York State Journ.,'* 1921, XXI, *p.* 138).—**T. Clarke** believes that true intestinal colic due to indigestion in the nursing infant is rare, and that for the intelligent handling of such cases the first essential is to disregard the maternal diagnosis. A very frequent cause of the condition is hunger. The trouble with the mother's milk is more frequently quantitative than qualitative. A second class of cases usually called colic by the family is that of otitis media. The third cause is old-fashioned spunk aggravated by bad training and bad habits.

J. ALLAN.

Diagnostic significance of abdominal pain in children (*'Brit. Med. Journ.,'* 1921, I, *p.* 1).—**R. Hutchison** divides cases of abdominal pain into three groups: (1) in the abdominal wall; (2) in the thorax; (3)

intra-abdominal. As examples of (1), (*a*) spinal caries is the commonest. Pain is referred to the epigastrium and may be present before deformity, but there may be slight prominence of the vertebræ, tenderness on percussion and some rigidity. (*b*) Lateral scoliosis in extreme cases. (*c*) Fibrositis affecting muscles of abdominal wall. Pain is felt at the insertion of the muscles into the ribs and iliac crests or in the aponeurotic sheaths of the muscles. Tenderness is experienced on sitting up in bed without using the hands. (*d*) Omental herniæ and undescended testis are also associated with pain. (*e*) Acute inflammation of or around the hip-joint. (*f*) Herpes zoster is accompanied by a superficial area of tenderness in the distribution of the affected nerves and by severe pain near the vertebral spines, and by absence of deep tenderness and rigidity on palpation. (2) Pneumonia simulates appendicitis, and is distinguished by alteration in pulse-respiration ratio, by absence of tenderness in the recto-vesical space, and by higher temperature. Pericarditis affecting the diaphragmatic surface may closely imitate appendicitis, the abdomen being rigid. (3) Acute catastrophic pain may be caused by (*a*) acute indigestion, which may be sudden and acutely painful with high temperature; (*b*) appendicitis, in which the pain usually starts in the epigastrium and is the first symptom, followed by vomiting, which does not persist; (*c*) by intussusception; and (*d*) by abdominal purpura. Chronic recurrent pain may be due to (i) ordinary colic, which occurs in spasms, is relieved by pressure, thus differing from appendicitis, and by passage of flatus; (ii) umbilical colic, which is referred to the umbilicus and may occur at any time, especially after or during meals, may be so severe as to double the child up and may be associated with sudden pain, vomiting being rare; (iii) entero-spasm, which is associated with membranous colitis and with mucus and membranes in the stools. There may be fever, and the contracted bowel may be felt in the left iliac fossa. Chronic intestinal obstruction is usually due to pressure by a band or a persistent Meckel's diverticulum. Caution should be exercised in diagnosing chronic appendicitis. Physical examination between the attacks reveals nothing, and X rays are not very helpful. Enlarged glands in the mesentery are frequently associated with appendicitis and cause pain in the right lower quadrant of the abdomen. Kinking of the ureter is due to a congenital abnormality of the renal vessels and causes attacks of periodic abdominal pain and vomiting. It is *not* associated with urinary symptoms. Ureteric calculus causes right-sided spasmodic pain and tenderness at or close to McBurney's point. CHRISTOPHER ROLLESTON.

Pathogenesis of cœliac disease (*'Arch. of Ped.,'* 1923, XL, *p.* 88).—**R. Miller** summarises important points in the disease as (1) failure to absorb fat, with excess of fat in the stools in the form of fatty acids, (2) enlargement of the abdomen, (3) retarded growth and development, (4) impaired appetite, (5) attacks of fever. The pancreas, the liver and the intestines have all been said to be at fault, but of the six recorded autopsies four showed no hepatic, pancreatic or intestinal lesion. The other two were atypical cases and in them fibrosis of the pancreas and intestine were found. Mackenzie Wallis, after the observation of 6 cases, states that definite evidence of pancreatic insufficiency is wanting. Further, in pancreatic disease the fat in the stools is present in the form of neutral fat, while in cœliac disease it is present in the form of fatty acids and soaps. Against Herter's theory of an enteritis is the fact that diarrhœa is not an invariable or

constant symptom of the disease. The withdrawal of fat from the diet is the main plank in modern treatment and proves that the disorder is a fat indigestion. Further, where severe diarrhœa does produce failure of fat absorption, the clinical picture is not that of cœliac disease. The pallor of the stools is due, not to an absence of bile pigments, but to their presence being masked by excess of fatty acid crystals, for if the liver's action were inhibited jaundice or hæmatoporphyrinuria would occur. In pancreatic steatorrhœa the stools are just as pale, although there is no interference with the passage or function of bile. Again, in two autopsies the gall-bladder contained coloured bile. On the other hand, bile-salts are found in smaller quantities and are absorbed from the intestine in conjunction with the fat. In cœliac disease it is possible that there is an excessive loss of bile-salts in the fatty stools. Effective fat absorption is thus prevented. In favour of this view are the facts that fat is efficiently split up into fatty acids and soaps, and that bile-salts are of value in treatment, reducing the amount of fat in the fæces, in one case from 57 per cent. to 26 per cent., and in another from 52 per cent. to 33 per cent.

CHRISTOPHER ROLLESTON.

The cœliac disease (*'Amer. Journ. Dis. Child.,'* 1923, xxv, *p.* 46).—**R. Taylor** alludes to R. Miller's paper on this subject (see BRITISH JOURNAL OF CHILDREN'S DISEASES, 1921, xviii, p. 11), and records some personal cases, in all of which the disease had been preceded by a long period of unsuitable feeding or a parenteral infection, such as pertussis or varicella, or both. Gastric achlorhydria was present in the five cases in which the stomach contents were examined. X rays in each case showed a dilated atonic colon. The liver was smaller than normal in every case. One case was complicated by Banti's disease, another showed leucin and tyrosin in the urine, and in another the duodenal fluid contained urobilinogen. Taylor recommends that the following principles should be followed in the treatment of the disease: (1) The use of sour milk and fruit acids in recognition of the achlorhydria; (2) a supply of readily absorbable carbohydrate primarily for its good effect on the liver; (3) restriction of the diet to proteins for months before adding starch and fats; (4) prevention of fæcal accumulation, which may undergo bacterial decomposition. J. D. ROLLESTON.

Non-diarrhœic type of cœliac disease (*'Lancet,'* 1923, i, *p.* 72).—**R. Miller** and **H. Perkins.**—The onset of this type is insidious and occurs at the same age as the diarrhœic variety, namely the second year of life. The abdomen is only moderately enlarged. The appetite is very poor. There may be retardation of growth, but more commonly only failure to gain weight. The stool is large but well coloured and not particularly offensive. The fat is in excess of the normal figure of 20–30 per cent., and is chiefly in the form of soaps. There is also a tendency to pyrexia. Convalescent cases of ordinary cœliac disease still show excess of fat in the form of soaps, even when the white diarrhœa has disappeared. Further, in true cœliac disease the stools vary in character from day to day, being sometimes furred and coloured and at other times pale and unformed. This depends upon the proportion of soaps present. When these are high the stool will be coloured. Treatment is on the same lines as for ordinary cœliac disease. The milk should be skimmed. This is best done by heating the milk to 200° F. for 20 minutes and then letting it stand. The cream thus clotted is

removed. Butter should be replaced by anchovy paste, potted meat, jams, suet and meat. Fats are not allowed. Eggs, lean meat, chicken, vegetables, bread and toast are allowed, and for pudding boiled rice, sandwich and sponge cake puddings. If the weight and the symptoms diminish on this form of diet a few scrapes of butter may be allowed and a little suet used in cooking. Kapsols, prepared by Allen & Hanbury, consisting of ½ gr. each of sodium tamocholate and glycocholate, can then be given. Neutral fat should not exceed 25 per cent. of the total fæcal fat. A greater proportion suggests failure of the pancreatic lipase. Such stools are soft, oily and very frequent, and the fat separates out as soft oily droplets. This condition is entirely different from cœliac disease. The pallor of the cœliac stool is due not to the absence of bile salts, but to their being masked by fatty acids. The author has seen catarrhal jaundice in cœliac disease, and again in pancreatic steatorrhœa the stools are pale, although there is no interference with the bile flow. CHRISTOPHER ROLLESTON.

Otology, Rhinology, and Laryngology.

Aural sepsis in infancy and childhood (*'Practitioner,'* 1922, CIX, *p.* 57).—**W. Stuart-Low** emphasises the importance of pulling the auricle downwards to obtain a good view of the infant's tympanic membrane. There may be no bulging of the membrane in acute otitis if drainage is possible into the naso-pharnyx, but there is always redness. There may be no pain or rise of temperature. The operation of incision of the drum should be done under chloroform. Glycerine and carbolic drops should be inserted in the ear and a pad of wool placed over the ear, but no meatal plug. The incision should extend from the lowest point behind the handle of the malleus right up and into the attic through the ventricular fold. Gonorrhœal otitis media is by no means rare and is sometimes associated with gonorrhœal ophthalmia. Protargol is equally effective in both. Profuse, thick, purulent and painless discharge is often caused by the tubercle bacillus. It ends in extensive destruction of the bone or meningitis. Facial paralysis is common in such cases, owing to the extension of the disease to the aqueduct and pressure on the nerve or to periostitis. The tubercle bacillus is very difficult to find in aural discharges. Under one year 50 per cent. of cases of purulent otitis are due to tubercle, and below that age 27 per cent. It may be a primary infection by direct contact, or through the blood-stream, or by the Eustachian tube. There may be mere infiltration of the mucous membrane or necrosis of the temporal bone with rapid caseation and destruction of the structures, even to exfoliation of the entire labyrinth. Good results are obtained by spraying first with the mitigated and then with the full-strength tincture of iodine into the ear. Syringing should not be undertaken, and peroxide of hydrogen should be avoided. Operation by curetting and removal of carious bone and by the complete mastoid operation is advisable if the disease be primary and not secondary to pulmonary tuberculosis. CHRISTOPHER ROLLESTON.

The importance of otitis in infancy (*'Bull. et Mém. Soc. Méd. des Hôp. de Paris,'* 1921, 3e sér,, XLV, *p.* 1326).—**M. Renaud** is convinced that otitis and suppuration in the petrous portion of the temporal bone play an important part in the pathology of the infant. In the course of 70 autopsies on infants whose deaths had been attributed to infantile diarrhœa he invariably

found extensive suppuration in the middle ear and petrous portion of the temporal bone. He concludes that suppuration in the middle ear and petrous portion of the temporal bone is the essential if not exclusive cause of death in infants, and that the presence of this condition renders spontaneous recovery impossible. He maintains that a systematic examination of the ears should be made in all sick infants, and that it is more important than examination of the chest, and perhaps even of the throat, since the latter, if infected, can be drained spontaneously, whereas the ear can only be drained by surgical operation. J. D. Rolleston.

Treatment of aural discharge in children (*'Lancet,'* 1923, i, *p.* 351).—**H. Barwell** advises that as soon as a child complains of earache the meatus be cleared of wax by gentle syringing with 1 in 60 carbolic lotion, and antiseptic drops of carbolic acid and 25 gr. glycerine to the ounce should be instilled four-hourly. Dry heat should be applied by means of an indiarubber hot-water bottle over a large pad of cotton-wool. When the drum bulges it should be incised and a narrow drain of gauze inserted, which is changed daily by the doctor. Pain and fever should cease within two days, but if these symptoms persist with tenderness over the mastoid, the antrum and mastoid cells must be opened. Symptoms of meningitis, delirium, retraction of the head and strabismus often subside after perforations of the drum. In infants there are often no localising symptoms. So that in all cases of unexplained pyrexia the ears should be examined. If at the end of three weeks the discharge still persists even though pain and tenderness have disappeared the antrum and mastoid cells should be opened. In chronic cases the discharge should be carefully mopped out and drops of boric acid 15 gr. and rectified spirits ℥j instilled. Small granulations should be touched with silver nitrate. The mastoid operation should not be performed until the child is over 12 years of age, unless the discharge is very foul. Adenoids and tonsils should be removed when the acute symptoms have subsided. Christopher Rolleston.

A case of latent intracranial abscess (*'Lancet,'* 1922, i, *p.* 992).—**M. Vlasto** and **S. Owen** describe a case in a boy, aged 8 years, who had been ailing since August 23, 1921, with pains in the head and vomiting. His temperature on August 29 was 102° F. and his pulse 140. He had bilateral otorrhœa with tenderness and swelling behind both ears. He was delirious, and head rigidity, retraction of the neck and Kernig's sign were present. Turbid but sterile cerebro spinal fluid was withdrawn on lumbar puncture. A diagnosis of double mastoiditis and early meningitis was made. The optic discs at this stage of the illness were normal. A double mastoid operation was performed on August 29. Pus was found in both antra and drainage tubes were inserted. For nine or ten days the condition remained unchanged. On September 3 erysipelas developed, followed by subcutaneous abscesses in various parts of the body. On September 14 definite improvement was noted, and by September 23 the temperature and respiration rate were normal, but the pulse was still rapid—100–112. On September 30 headache and vomiting recurred. The temperature was subnormal and the pulse-rate 60. He was drowsy and very wasted. The optic discs were choked, the right more than the left, and there was general hypotonicity. An exploring operation was performed but no abscess was found, although the cerebro-spinal fluid contained Gram-positive diplococci.

Marked improvement followed and the pulse increased in frequency, but the temperature remained subnormal and headache and vomiting occurred every second day. The papillœdema was subsiding, and repeated examinations of the nervous system were negative. The stage of convalescence was again interrupted on November 12 by headache, vomiting, drowsiness and slow pulse. There was an increase in the papillœdema, the swelling of the left disc being now greater than the right. There was greater hypotonicity of the left arm, disinclination to move it, but no definite paralysis. Both posterior fossæ of the skull were explored on October 1 with a negative result. The patient died suddenly on October 20. At the necropsy a large abscess was found occupying the right middle and anterior fossæ. The abscess extended to within an inch of the superior longitudinal sinus; the pus was situated between the dura and the pia arachnoid and showed Gram-positive diplococci and staphylococci. CHRISTOPHER ROLLESTON.

Nasal obstruction in infants during the first year of life (*'Lancet,'* 1922, I, *p.* 697).—**A. Jones,** in a communication to the Manchester Medical Society, said the commonest causes of this condition were: (1) Congenital occlusion of the nostrils; (2) the temporary presence of mucous secretion in the nose and engorgement of the nasal mucosa during the first few days of life; (3) congenital syphilis; (4) mongolism; and (5) adenoids—the commonest. The symptoms usually seen were: inability to suckle efficiently, snoring and suffocation attacks, persistent cough, and nasal discharge and convulsions. As to the differential diagnosis, congenital occlusion of the nostrils was very rare. A probe would reveal the situation and completeness of the obstructing web. Temporary engorgement of the nasal mucosa was at once relieved by a weak adrenalin spray. Congenital syphilis usually presented constitutional symptoms, the Wassermann reaction being positive. A Mongol was recognisable by his typical appearance, and adenoids were diagnosed by excluding the other causes. The case should be dealt with according to the cause. In adenoid cases surgical removal was simple, efficient, free from risk and gave excellent results. J. ALLAN.

Hypertrophic rhinitis in the child (*'Thèses de Paris,'* 1920–21, *No.* 479).—**P. Gand** states that hypertrophic rhinitis is met with in children of both sexes. According to Lermoyez it is twice as common in boys as in girls. The predisposing causes are damp, sudden change of weather and overeating. The local factors are adenoids and narrowness of the nasal fossæ. Local and general disturbances result. The local disturbances are partial and complete, permanent or transitory, nasal obstruction and an acute or chronic rhinorrhœa. The general disturbances are of three kinds: (1) Infective manifestations, such as laryngitis, tracheitis and bronchitis. (2) Trophic changes, such as deformity of the thorax, mal-development of the facial skeleton, mental troubles and aprosexia. (3) Reflex disorders of the cardiac or respiratory system and various spasmodic conditions, such as asthma or epilepsy. Three clinical types of hypertrophic rhinitis may be described: (1) A purely congestive type which subsides under treatment with adrenalin 1 in 20,000; (2) a hyperplastic type which requires a stronger solution of adrenalin; and (3) a myxomatous type due to polypoid degeneration of the turbinates. Treatment consists in cauterisation with chromic acid after local anæsthesia with cocaine. Thirteen personal cases are recorded. J. D. ROLLESTON.

Nasal diphtheria and foreign bodies (*'Lancet,'* 1922, I, *p.* 914).—**J. Keen** and **T. A. Carson** report two cases in a girl, aged 7 years, and in a boy, aged 9 years, respectively. In both there was persistent unilateral nasal discharge, and swabs, taken repeatedly, were reported positive to K.L.B. In each case a shoe-button was removed from the nostril, after which the condition cleared up. The authors suggest the advisability of an exhaustive search for a foreign body in all cases of unilateral nasal discharge even in the face of a positive report, and they incline to the view that a condition of persistent nasal diphtheria does not exist unless caused by a foreign body.

J. ALLAN.

The treatment of coryza in infants (*'Lyon Méd.,'* 1921, CXXX, *p.* 850).—**P. Chatin,** recognising the gravity of certain coryzas in young infants, is of opinion that the application to them of most of the methods of treatment recommended for adults is highly dangerous. For instance, menthol, which is an inoffensive preparation in the adult, may produce in an infant spasm of the glottis and fatal consequences. As to mechanical intervention such as douches, spraying, inhalation and insufflation, these cannot be used on an infant. The author recommends a method of treatment through the lacrymal ducts which has the following advantages; the deep application of a liquid antiseptic through a natural passage which is always permeable without risk of injuring the inflamed mucous membrane; a continuous instillation by drops through the lacrymal apparatus with the admixture of the antiseptic with the lacrymal secretion which has for its normal function the moistening of all parts of the nasal fossæ. He uses for this purpose a solution of 10 per cent. argyrol in water, which is introduced into both eyes of the infant two or three times a day in mild and more often in serious cases. At the end of two or three days marked improvement takes place, and the method is free from all disadvantages and is well tolerated.

VINCENT DICKINSON.

The diseased tonsil (*'New York Med. Journ.,'* 1921, CXIV, *p.* 649).—**M. S. Ittelson** considers that tonsils are frequently removed without adequate reason, being often affected by causes which lie largely outside the tonsil itself, such as pathological conditions in the nose, mouth sinuses, intranasal operations, or outside infection through the air or food. In removing diseased tonsils, what we remove is the end-product without removing the basic cause, and the result often is a hypertrophy of the lymphoid tissue in the neighbourhood, viz. in the pharynx. The infra-tonsillar nodules extending down to the lingual tonsil and to the epiglottis are left behind and often continue to act as a source of infection. Ittelson points out that the crypts of the tonsils contain *débris* and organisms from soon after birth which have no bad effect on the general health. He admits that the literature is full of the brilliant results which have followed tonsillectomy, but considers that in the interests of the patients the present indications for operation should be carefully revised, and then unnecessary operations would be fewer.

J. PORTER PARKINSON.

Vincent's bacillus in affections of the oral cavities (*'La Pediatria,'* 1921, xxix, *p.* 539).—**F. de Angelis** found that out of about 22,000 children seen at the clinic at Naples during 1914–20, there were 143 cases of affections of the oral cavities due to fuso-spirillary organisms. They were never met

with in infants under a year, but occurred most frequently from 2 to 6 years of age. The gums and cheek were the parts most often affected, the tonsils only in 6·2 per cent. Diagnosis was easy from the constant presence of ulceration, easily bleeding necrotic changes and marked fœtor. The most efficacious treatment was in douching with hydrogen peroxide and subsequent use of a calcium chloride powder. Intravenous injections of novarsenobenzol were also advantageous. VINCENT DICKINSON.

Tonsillectomy in infancy and childhood (*'Arch. of Ped.,'* 1923, XL, *p.* 20).—**E. M. Tarr** from June, 1919, to June, 1922, removed 800 pairs of tonsils in private practice in addition to several hundreds in hospital. There were no deaths, no otitis or mastoiditis, no lung abscess, and only 3 bronchopneumonias. In all these cases gross infection of the tonsils was the prime indication for the operation. Ninety per cent. of the operations were done with a modified Sluder instrument. Tarr recommends that the coagulation time should be determined in every case, as this will do much to prevent hæmorrhage. J. D. ROLLESTON.

An analysis of the end-results of tonsillectomy and adenoidectomy (*'New York Med. Journ.,'* 1922, CXVI, *p.* 142).—**S. A. Mauner** and **S. Z. Orgel**, as a result of the study of 100 cases, come to the following results: These operations are of advantage in tonsillitis and chronic otitis media, also in half the cases of colds, mouth-breathing, malnutrition and anæmia. More advantageous results are obtained where a complete removal of tonsils and adenoids was performed. If the only indications are that the tonsils are visible to the naked eye and no symptoms are present, in children who have not had an illness involving the tonsils or adenoids, there is no reason for surgical interference. J. PORTER PARKINSON.

Tonsil enucleation and the tonsil enucleator (*'New York Med. Journ.,'* 1921, CXIV, *p.* 639).—**C. B. Meding**, after reviewing the changes in methods of removal of tonsils, has used a modification of the Beck-Schenck tonsillectome since 1914, with most successful results. Two hundred analyses of the organisms found showed them to be exceedingly varied, so that no classification could be made. His rule is to remove all tonsils, whether enlarged or not, if they cannot keep clean, and whether or not there be any enlargement of the cervical glands. The writer has devised a stronger and simpler form of the Beck-Schenck instrument, which has a flat and rigid steel loop which grips the tonsil concentrically and peels the capsule from its bed. He uses ether alone or preceded by gas up to surgical anæsthesia for adults, and less for children. He has used this method for 10,000 cases and never had a severe hæmorrhage or a scarred pharynx or torn palate.
J. PORTER PARKINSON.

Tonsillectomy in children with endocarditis and frequent tonsillitis (*'New York Med. Journ.,'* 1922, CXVI, *p.* 145).—**M. H. Kaiden** quotes two cases where the removal of tonsils greatly benefited patients with acute rheumatism of probable tonsillar origin. He quotes Osler and McCrae and Fleischner, who advocate this treatment. The points of importance are—history of frequent sore throats, a red "corned beef" appearance of the anterior pillars indicating a diseased tonsil, the exudation of a cheesy

whitish material on pressure on the tonsil, and enlarged tonsillar lymph-glands at the angle of the jaw. J. Porter Parkinson.

Reinfection of tonsillar remnants after tonsillectomy (*'New York Med. Journ.,'* 1923, cxvii, *p.* 221).—**J. A. Glassburg** quotes three cases where, after incomplete tonsillectomy, tonsillitis has recurred. One was septic and two were rheumatic cases, in one of which a peritonsillar abscess was present. Removal of the remaining remnants of tonsil prevented further recurrences. He quotes these cases as an argument for complete removal of the tonsils. J. Porter Parkinson.

Tonsillar and adenoid tissue under X-ray treatment (*'New York Med. Journ.,'* 1921, cxiv, *p.* 646).—**W. G. Herrman** relates his experience in the X-ray treatment of diseased tonsils and adenoids. The head and shoulders of the patient are covered with lead except for an opening two to three inches square over the site of the tonsil. The time of exposure varied, but never more than one skin unit filtered was given. Six to eight treatments at two weeks' intervals sufficed, and no redness of skin or other complication occurred. The tonsillar tissue shrunk and the crypts lost their contents. The cases that had strepto- or staphylococci in the tonsils before treatment became sterile. The chief disadvantage of the treatment is the time necessary to produce the results. J. Porter Parkinson.

Treatment of enlarged tonsils by X rays (*'Paris méd.,'* 1923, i, *p.* 109).—**T. Nogier** states that some forms of tonsillar enlargement are more suitable than others for this treatment. In true hypertrophy, in which all the anatomical elements of the tonsil are more or less enlarged, a hard and a soft form is described. The soft form, which is more frequent in children than in adults, is most amenable to treatment, while in the hard form the results of treatment are very slight owing to the abundance of sclerotic tissue. In the lacunar type of tonsillar enlargement the crypts and follicles become distended by the products of secretion, giving rise to a pseudo-hypertrophy. In such cases X rays produce only slight improvement. Five units are applied to each tonsil once a month. The immediate results are manifested about a fortnight after the first application, and consist in a marked and progressive reduction in size and change in colour of the tonsils from a bright red to a pale pink, while the tonsillar surface, which was previously granular, becomes smooth. The functional symptoms also diminish in intensity, respiration becomes easier, snoring in sleep ceases, the voice becomes clearer, pain in swallowing disappears and the general health improves. In Nogier's cases these results were maintained for several years. The advantages of X-ray treatment of enlarged tonsils over other methods are that it is simple, painless, and does not expose the patient to the risks of hæmorrhage and infection. It is the only method that can be used in cases of hæmophilia or Bright's disease. J. D. Rolleston.

Congenital laryngeal stridor complicated by perilaryngeal abscess (*'Le Nourrisson,'* 1922, x, *p.* 187).—**G. Blechmann** and **Peignaux** report a fatal case in a female infant, aged 7 months, who presented all the signs of congenital laryngeal stridor. Enlargement of the thymus was excluded by radioscopy. As the dyspnœa increased and there was considerable difficulty

in swallowing inoculation was performed, with great relief, but death from broncho-pneumonia took place a few days later. The autopsy confirmed the absence of any enlargement of the thymus, and showed not only the existence of the malformation in the larynx characteristic of congenital stridor, but also an abscess external to the left side of the larynx from which various organisms, mainly streptococci, were cultivated.

J. D. Rolleston.

Direct laryngoscopy in children (*'Nederl. Tijdschr. v. Geneesk.,'* 1922, i, *p.* 193).—**E. Huizinger** states that during the last 12 months Chevalier Jackson's method of direct laryngoscopy has been used in the Groningen Nose and Throat Clinic. The child is placed on a flat operating table, the head being fixed in a position of moderate extension by one assistant; too great extension interferes with laryngoscopy and may be dangerous. A second assistant keeps the arms to the chest and a third holds the legs. No anæsthetic is required. Huizinger records cases of foreign body, chronic laryngitis and laryngeal polypus successfully diagnosed and treated by this method.

J. D. Rolleston.

Correspondence.

To the Editor of The British Journal of Children's Diseases.

Hereditary Tylosis.

Dear Sir,—In his interesting article on "Hereditary Tylosis" Dr. J. D. Rolleston* refers to the condition on the Island of Meleda having been at first described as a kind of leprosy. It is noteworthy that in England, as was pointed out by Dr. G. Pernet, a case of congenital (though apparently not familial) tylosis was regarded as a form of leprosy by Gilbert White, the author of 'The Natural History of Selborne.' In a letter to the Honourable Daines Barrington, dated from Selborne, January the 8th, 1778, he wrote as follows:

"There was in this village several years ago a miserable pauper, who, from his birth, was afflicted with a leprosy, as far as we are aware of a singular kind, since it affected only the palms of his hands and the soles of his feet. This scaly eruption usually broke out twice in the year, at the spring and fall; and, by peeling away, left the skin so thin and tender that neither his hands or feet were able to perform their functions; so that the poor object was half his time on crutches. . . . His habit was lean, lank, and cadaverous. In this sad plight he dragged on a miserable existance, a burden to himself and his parish, which was obliged to support him till he was relieved by death at more than thirty years of age. The good women, who love to account for every defect in children by the doctrine of longing, said that his mother felt a violent propensity for oysters, which she was unable to gratify; and that the black rough scurf on his hands and feet were the shells of that fish. We knew his parents, neither of which were lepers; his father in particular lived to be far advanced in years."

* British Journal of Children's Diseases, 1923, xx, p. 16.

The case was doubtless one of an acroteric nævus of the tylosis kind, and, as usual in cases of nævus of all kinds, was popularly accounted for as the result of "maternal impression."

I am Sir,
Yours faithfully,
April the 7th, 1923. F. PARKES WEBER.

To the Editor of THE BRITISH JOURNAL OF CHILDREN'S DISEASES.

DEAR SIR,—I was much interested in Dr. J. D. Rolleston's paper on Hereditary Tylosis. There are, I think, in the island of Stronsay, Orkney, Scotland, at least two families which show good examples of this disease—the only cases I have ever seen. I have not been there for some years, but, as far as I can remember, cases occur in at least three generations, and if enquiries were made, could possibly be traced in five generations. There were two large families, in one of which the females suffered, and in the other the males. Only one parent was affected in each family, and if I remember aright the disease was transmitted to the children of the opposite sex by the affected parent. The families were large and in poor circumstances, like the case described by Dr. Rolleston. Some of the children, no doubt, have married now, and may have transmitted the disease to their children. Until I read Dr. Rolleston's paper I had never seen a similar case nor met with a doctor who had.

I am,
Yours faithfully,
C. B. BROWNLIE, M.B., Ch.B.

NIXON VILLA,
MERTHYR VALE,
GLAMORGANSHIRE;
May 28th, 1923.

Reviews.

BULLETIN OF THE BUFFALO GENERAL HOSPITAL. Vol. i, No. 1. January, 1923. Published by the Board of Trustees of the Hospital. Edited by a Committee of the Staff.

WE offer our cordial congratulations to the Editorial Committee on the first number of this new journal, which contains thirteen papers on unusual and instructive cases. Those likely to be of special interest to our readers are: "A Report of a Case of Labour in an Achondroplasic Dwarf with Delivery by Cæsarian Section of an Achondroplasic Child," by Elmer A. D. Clark and E. C. Koenig; "A Report of a Case of Rheumatism in an Adult, with Biopsy and a Brief Review of the Literature," by Hobart A. Reimann; and "Congenital Stenosis of the Œsophagus: A Case Report of its Successful Treatment," by Karl F. Eschelman. J. D. R.

Monatsschrift für Kinderheilkunde. Band xxv. Festschrift für Adalbert Czerny. Leipzig: F. C. W. Vogel, 1923. Pp. 693.

The present volume, which is the first 'Festschrift' dedicated to a pædiatrist since that published in honour of the late Professor Baginsky in 1913 (see British Journal of Children's Diseases, 1913, x, p. 382), has been offered to Professor Adalbert Czerny, of Berlin, by his friends and pupils on the occasion of his sixtieth birthday. It contains 59 articles, which are contributed not only by representatives of Germany, Austria and Hungary, but also by pædiatrists in the United States, Italy, Switzerland, Norway, Sweden, Denmark and Poland. Ten more papers intended for the 'Festschrift,' but despatched too late, are to appear in the May issue of the journal. The subjects discussed cover the whole field of pædiatrics, including experimental medicine, metabolic studies, infant feeding, acute infectious diseases, tuberculosis, syphilis, diseases of the alimentary system, diseases of ductless glands, nervous and mental diseases, diseases of nutrition, skin diseases, and therapeutics. J. D. R.

Premature and Congenitally Diseased Infants. By Julius H. Hess, M.D., Professor of Pediatrics, University of Illinois. Pp. 397. Illustrations 189. London: J. & A. Churchill, 1923. Price 18s.

Dr. Hess garners a somewhat neglected patch of medicine in gathering together a large number of observations on the premature infant, and, by this means, providing a valuable and interesting work of reference of great practical value. The first part is devoted to ætiology, physiology and pathology, and contains a mass of useful data and tables. In the second part the methods of nursing and feeding are fully considered and clearly expounded. A special word of commendation must be given to the detailed description of wet nursing and its management. The third part consists of nine chapters on general and special diseases. These are adequately described, and obviously the size of the book is insufficient to include every disorder which may occur in these infants. In the fourth part the prognosis and future of the premature infant are duly weighed.

We can congratulate Dr. Hess on producing an excellently written and well-balanced work, thoroughly up-to-date, sure to prove useful to those interested in these infants and those in want of a work of reference on this particular subject. In a sense it may be regarded as redundant to discuss prematurity independently of ordinary infancy, seeing that the premature infant differs little from the delicate infant in its general requirements as to management and nursing. For this reason, no doubt, Dr. Hess includes congenitally diseased infants. These he defines as the *weaklings*, and classifies as the *congenitally diseased* or *debilitated*. The numerous illustrations add greatly to the interest of the book, though some of them appear to have little connection with the subject. The publishers deserve much commendation on the format of the book, the clearness of the printing and the absence of misprints. In view of the large number of tables and engravings the price is decidedly moderate. E. C.

Anæsthesia in Children. By C. Langton Hewer, M.B., B.S.Lond., M.R.C.S.Eng., L.R.C.P.Lond., Assistant Anæsthetist to St. Bartholomew's Hospital, etc. Pp. 111. 31 illustrations. London: H. K. Lewis & Co., Ltd., 1923. Price 4s. 6d. net.

This small book should be given a cordial welcome, as it deals with a subject which has in the past received far too little attention. The author

has endeavoured to indicate the special features of anæsthesia applicable to children. He very aptly points out that the average student who qualifies at a general hospital has usually had very little experience in anæsthetising children, and leaves under the vague impression that they only want a whiff of anæsthetic and they will be all right. No greater mistake could be made, and the uninitiated will soon discover that the child presents a difficult problem, requiring quite as much skill to anæsthetise as does the adult, if not more.

The opening chapter contains some useful hints on the preparation and after-treatment of the patient, and in Chapter II some general principles are discussed. The next chapter is devoted to short anæsthesias in children, and includes information regarding the exhibition of nitrous oxide and ethyl chloride. Chapter IV deals with ethanesal, a comparatively new anæsthetic, and gives a short account of the research leading to its preparation, its composition, action and method of administration. Anæsthesia with chloroform, ether and mixtures is next reviewed, and this is followed by chapters on special methods of general anæsthesia and anæsthesia in certain common operations in children. In the final chapter local analgesia and spinal analgesia are considered, but for obvious reasons these have only a limited sphere of usefulness in children. Dr. Hewer is to be congratulated on his concise and practical presentation of the subject of anæsthesia in children. The general principles enunciated are sound, though there may be differences of opinion on certain details. This little book, which is full of common-sense teaching, should fill a long-felt want, and can be cordially recommended to residents in children's hospitals and to junior practitioners.

J. A.

A Code of Rules for the Prevention of Infectious and Contagious Disease in Schools. London: J. & A. Churchill, 1923. Price 2s. 6d.

This work begins with the prescribed periods of isolation of infected scholars. A section on the general sanitary arrangements follows, and it is curious to observe in the light of modern knowledge the importance assigned to drains. Although carriers are mentioned in connection with diphtheria, they are not definitely alluded to in cases of cerebro-spinal fever. Paratyphoid fevers and dysentery are not even mentioned—a strange omission in view of recent epidemics of the last-mentioned disease in schools, and considering that the hygienic rules for the dairy are not omitted.

To the Schick test only cursory reference is made, and no directions are given as to its application.

In Appendix B on the notification of infectious disease a very imperfect list is given of the diseases of which the medical officer of health has to be informed. Pneumonia should at least have been included, and surely tuberculosis.

The paragraph announcing the fact that more than one medical man can notify the same case might well have been omitted.

Excellent rules are given as to the disinfection of rooms, linen, discharges from the patient, and infected crockery.

C. R.

Les Cardiopathies au cours de la Chorée de Sydenham. By Dr. Jean Vidal. Paris: A. Maloine & Fils, 1923. Pp. 86.

This thesis is devoted to the discussion of those problems of perennial interest, the relationship between chorea and the rheumatic infection, the

structural character of the cardiac changes in chorea and the clinical diagnosis of the minor heart affections. These subjects are dealt with fully and with a wealth of careful detail. The results of the author's investigation are fully in line with the commonly accepted English views, and it is of particular interest that he reaches the conclusion so insisted upon by the late Dr. D. B. Lees in this country that for the recognition of the earliest changes in the myocardium percussion is a method of greater value than auscultation. The author is to be congratulated upon the care with which he has dealt with the clinical material at his disposal and upon the way in which he has presented his subject. The references to the English literature dealing with the points discussed might well have been increased in number.

R. M.

La Gymnastique Respiratoire, et la Gymnastique Orthopédique chez soi. Par le Docteur Louis Lamy, Assistant d'Orthopédie à l'Hospice des Enfants Assistés. Paris: J. B. Baillière et fils, 1923. Pp. 128. Price 6 fr.

In this elementary book on the value of respiratory and other exercises the author points out the necessity for *individual* training in backward children, more especially those with nasal obstruction, bad stance and so forth.

Individual exercises lead to self-control and self-respect, and can then be followed by *class* exercises, thereby stimulating healthy competition.

Like English authors, M. Lamy attaches due importance to muscle rest, and gradually introduces simple apparatus.

Remedial exercises are dealt with in full, more especially with regard to the treatment of kyphosis, scoliosis and flat-foot, with due reference to the methods of Klapp, and the use of special celluloid jackets to maintain the correction and benefit of exercises.

All these exercises are followed by massage.

The book is well illustrated with simple shaded drawings, and its keynote is simplicity, with a keen desire to obtain the co-operation of patient, parent and instructor.

B. W. H.

Diagnostik und Therapie der Kinderkrankheiten, mit Speziellen Arzneiverordnungen für das Kindesalter. Ein Taschenbuch für den Praktischen Arzt. Von Prof. Dr. F. Lust. Dritte neubearbeitete Auflage. Berlin and Vienna: Urban & Schwarzenberg, 1923.

This book attempts to cover much ground and compress the discussion within the limits of a "pocket-book." In consequence we doubt whether all the statements, short, dogmatic, and written in the language of the telegraph form, will be intelligible to those who are not already very familiar with the subject. As a means of revising knowledge and as a store of therapeutic tips and wrinkles, in a form which makes reference easy, the book has its value. It makes no attempt at originality, but is accurate, and the limited space at the author's disposal has been equitably distributed between the subjects. As in almost all German text-books, the chapters upon breast-feeding are clearly and fully written, and the statements made are, we believe, essentially true. The treatment of rickets is very different from that which it would receive in an English text-book at the present day—an

excessive intake of cow's milk is regarded as the chief ætiological factor—and the same may be said of infantile scurvy.

The treatment recommended in diabetes mellitus appears antiquated, and is not very clearly described. Intramuscular injection of milk is recommended in various conditions, such as the nocturnal excitement following on encephalitis lethargica, furunculosis and gonorrhœal conjunctivitis.

As indicated by the title of the book, space is mainly directed to diagnosis and treatment. In the earlier pages a paragraph is sometimes introduced with the heading "Prognosis," but often the want of any statement as to the recovery rate appears a serious drawback. Anyone seeking information as to the relative mortality, to take a few conditions at random, of noma and thrush, pleural effusion and empyema, retro-pharyngeal abscess and cervical adenitis would not find himself much the wiser.

The author regards "enucleation of tonsils" as an operation to be undertaken only with the greatest reluctance, and to be avoided before the age of ten. His reason given is the fear of hæmorrhage.

There are no illustrations, no charts, no personal experiences, and few expressions of opinion. H. C. C.

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